

Pediatric ECG Interpretation: An Illustrative Guide

The accompanying CD

This CD has been prepared for the reader's use with three purposes in mind:

- 1** as a self-evaluation tool for interpretation of ECGs
- 2** as a teaching reference for Cardiology fellows, residents, and house staff
- 3** as an invaluable resource for the Emergency Room physician or pediatrician who might obtain an ECG on a pediatric patient.

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Pediatric ECG Interpretation: An Illustrative Guide

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Introduction

Pattern recognition is an important learning tool in the interpretation of electrocardiograms (ECGs). Pediatric practitioners generally receive limited exposure to reading ECGs until faced with a patient with an arrhythmia or structural heart disease. The ability to clearly distinguish an abnormal ECG pattern from a normal variant in an emergency situation is an essential skill, but one that many physicians feel ill-prepared to confidently utilize. This atlas is directed at medical students, housestaff, and practitioners with limited background in pediatric cardiology.

In this atlas, we illustrate many of the ECG patterns a pediatric practitioner is likely to encounter. Normal variations of ECGs in children of different ages are presented, followed by examples of ECG abnormalities such as ventricular hypertrophy and atrial enlargement. ECGs of common forms of acquired heart disease are presented, followed by typical ECG examples of congenital heart disease. Arrhythmias are presented in sections on bradycardia and supraventricular and ventricular tachycardia, with a basic section on pacemaker ECGs. Simple techniques used to interpret mechanisms of arrhythmias are described, as the nonspecialist in cardiology or electrophysiology may not have a readily accessible resource for these ECG examples.

Pattern recognition in ECG interpretation is not intended to replace an understanding of the genesis of the ECG, but to complement the basic skills of ECG interpretation. Thus, the reader of this atlas is presumed to have mastered one of the many excellent texts on the basics of ECG interpretation.¹⁻⁵ This atlas relies on previously accepted norms for interpretation of pediatric ECGs.⁶ Other ECG norms obtained using computer-automated voltage amplitudes at sampling rates higher than in older ECG machines have been recently published.⁷ These data suggest some gender and ethnic variations in pediatric ECG norms likely exist, and may provide the basis for establishing new standards for abnormal values. Where helpful, we have provided limited references for those interested in further details on certain topics. With this approach, we hope that the reader will find ECG interpretation in children an enjoyable challenge.

ECG Interpretation

Interpreting ECGs involves a sequential analysis of each component of the tracing: **rate, rhythm, axis, intervals, morphology, and chamber hypertrophy and enlargement.**⁵ Reference values for normal age-related ECG measurements for pediatric patients are found in Appendix 1, modified from Davignon *et al.*⁶

Heart Rate: Normal heart rates on 12-lead ECG vary significantly with the age of the child. In the newborn, heart rates by ECG range from 90 to 170 bpm. The average heart rate increases slightly after the first week of age, to 105 bpm, and by 1 month of age may be 120 to 180 bpm. After the neonatal period, the heart rate drops gradually with age, ranging from 75 to 140 bpm by 3–5 years of age. By adolescence, the normal heart rate ranges from 65 to 120 bpm. Heart rates measured by ambulatory ECG (Holter) monitoring are significantly different than those recorded on the resting 12-lead ECG, reflecting the child’s level of activity.

Rhythm: The relationship of the P wave to the QRS complex is analyzed to determine the cardiac rhythm. The P wave of normal sinus rhythm is normally smooth and upright in leads I, II, III, and aVF, and biphasic in lead V1. In normal sinus rhythm, a P wave precedes each QRS complex, with a constant P–QRS relationship, and the heart rate falls in the normal range for age.

Automatic (ectopic) atrial rhythm is characterized by an abnormal P wave axis and/or morphology. *Primary atrial tachycardia* (Table 1, Classification of SVT) is an arrhythmia arising within the atria, such as atrial flutter or atrial re-entry, and is characterized by an abnormal P wave axis and/or morphology, with an abnormal atrial rate. The degree of atrioventricular conduction may vary from 1:1 to 2:1 or 3:1, or higher degrees of block in the presence of medications.

In the presence of tachycardia, one attempts to identify the mechanism of tachycardia by analysis of the QRS–P relationship.^{8,9} Retrograde P waves are frequently identified in supraventricular tachycardia, with a 1:1 ventriculoatrial or QRS–P relationship. A P wave buried in the QRS or at the end of the QRS complex is commonly seen in *atrioventricular nodal re-entry tachycardia*. A

Table 1: Classification of Supraventricular Tachycardia

• Supraventricular tachycardia utilizing accessory connections
Orthodromic reciprocating tachycardia (ORT)
Permanent form of junctional reciprocating tachycardia (PJRT)
Antidromic reciprocating tachycardia
Atrial tachycardia with antidromic conduction
Pre-excitation variants
• Atrioventricular nodal tachycardia
AV nodal re-entry tachycardia (AVNRT)
Junctional (automatic) tachycardia
• Primary atrial tachycardia
Sinus tachycardia
Atrial flutter
Atrial re-entry tachycardia
Automatic atrial tachycardia
Multifocal atrial tachycardia
Atrial fibrillation

negative P wave following the QRS complex may be seen with *orthodromic reciprocating tachycardia* utilizing an accessory connection (e.g., the Wolff–Parkinson–White syndrome). In orthodromic reciprocating tachycardia, the QRS–P interval is greater than 0.07 sec, in contrast to atrioventricular nodal tachycardia, where the QRS–P interval is typically less than 70 msec.

Wide QRS tachycardia may be due to supraventricular tachycardia conducted to the ventricles with aberrancy (bundle branch block), atrial tachycardia conducted antegradely via an accessory atrioventricular connection (such as atrial fibrillation in the presence of Wolff–Parkinson–White syndrome), or ventricular tachycardia. Knowledge of the presence of pre-existing bundle branch block or pre-excitation may allow comparison of the QRS morphology in sinus rhythm and during tachycardia to establish the mechanism. The QRS–P relationship is analyzed. The ECG diagnosis of ventricular tachycardia is suggested by ventriculoatrial dissociation, the presence of fusion beats, a leftward QRS axis, and marked QRS duration prolongation greater than the 98th percentile for age. Criteria useful for differentiating ventricular tachycardia from supraventricular tachycardia with aberrancy have been summarized in Appendix 2.^{9–11}

Axis: The frontal plane axis of the P wave, QRS complex, and T wave is analyzed sequentially. Sinus rhythm is characterized by a P wave axis usually between +30 and +90 degrees (upright P wave in leads I and aVF). A left atrial rhythm shows a P wave axis between +90 and +270 degrees (negative P wave in leads I and V6), and a low right atrial rhythm exhibits superior axis deviation of the P wave (upright P in lead I, negative in aVF).

The QRS axis shifts significantly with age. The neonate has a QRS axis typically between +60 and +210 degrees, with gradual shifting to the left with development. By 1–5 years of age, the QRS axis is +10 to +110 degrees. Variations in QRS axis may be due to ventricular hypertrophy or abnormalities of ventricular conduction (bundle branch block, ventricular pre-excitation).

The T wave axis should generally correspond to the QRS axis, known as QRS–T concordance. Greater than a 90 degree difference between the QRS and T wave axis, or QRS–T wave discordance, may reflect myocardial injury or strain.

Intervals: The PR, QRS, and QT intervals are measured individually, and are compared to age-related normal values. A normal PR interval is shorter in a child than in an adult. Normal PR intervals in the neonate range from 0.08 to 0.16 sec. The normal PR interval lengthens gradually with age, so that by 12–16 years of age, the normal PR interval is 0.09–0.18 sec. A short PR interval with a delta wave (slurred onset of the QRS complex) may be seen with manifest pre-excitation, or Wolff–Parkinson–White syndrome. The initial delta wave polarity may be helpful in determining the location of the accessory atrioventricular connection; one schema is included in Appendix 3.¹²

The QRS duration is typically shorter in a young infant than in an adult. Under 4 years of age, the QRS duration is less than 0.09sec, and less than 0.10sec up to 16 years of age. By late adolescence, the QRS duration should be less than 0.11 sec. Longer QRS durations are associated with abnormal intra-ventricular conduction (bundle branch block, myocardial injury, electrolyte disturbances, ventricular pre-excitation) or cardiac arrhythmia (ventricular tachycardia).

The QT interval, as measured from the onset of the Q wave to the end of the T wave in lead II, should be corrected for heart rate (QTc). In general, the normal QTc is less than or equal to 0.44sec. In the first several days of life there may be transient prolongation of the QTc, which should normalize after the first week of life. Prolonged QT intervals may be seen with congenital long QT syndrome, abnormal ventricular depolarization (bundle branch block), cardiomyopathy, or metabolic or electrolyte abnormalities.

Morphology: The appearance of the P wave, Q wave, QRS complex, and T waves is analyzed sequentially. Notching or prolongation of the P wave may indicate an ectopic atrial rhythm or other atrial tachycardia, atrial enlargement, or intra-atrial conduction abnormality.

Q waves are normally seen in the inferior leads II, III, aVF and lateral leads V5–V6. The presence of a Q wave in V1 is abnormal at any age, and may reflect right ventricular hypertrophy, ventricular inversion, myocardial infarction, left bundle branch block, or pre-excitation pattern. Pathologic Q waves are characterized by abnormally deep or wide Q waves. Deep Q waves in leads I, aVL, and the left precordium may be seen in infants with anomalous origin of the left coronary artery, reflecting a pattern of myocardial infarction. Deep Q waves in inferior and lateral leads may be seen in left ventricular hypertrophy.

Delays in ventricular conduction manifest as a prolonged QRS complex, usually with abnormal QRS morphology and axis. *Bundle branch block* is identified by QRS duration greater than normal for age (see above), and in children is usually due to surgery for structural heart disease, or cardiomyopathy. *Right bundle branch block* is characterized by an rsR' or rR' pattern in lead V1, with a broad, slurred S wave in leads I and V6. *Left bundle branch block* is identified by a tall, notched R wave in V6, with a broad slurred QS complex in lead V1.

The ST segment and T waves reflect ventricular repolarization. A normal ST segment is less than 1 mm above or less than 0.5 mm below the baseline. The T wave axis should be concordant with the QRS axis in most leads. In children, tall T waves in the mid and lateral precordium are often seen; in general, the T wave amplitude should be less than 10 mm in the precordial leads. Abnormal ST segments and T waves may reflect a normal variation such as early repolarization, or indicate pathology such as myopericarditis, metabolic/electrolyte imbalances, hypertrophy, cardiomyopathy, or long QT syndrome.

Chamber hypertrophy and enlargement: Atrial enlargement can be determined by ECG only when the patient is in sinus rhythm. The ECG cannot be used to reliably diagnose ventricular hypertrophy in the presence of bundle branch block, ventricular pre-excitation, paced ventricular rhythm, or ventricular arrhythmia.

Right atrial enlargement is characterized by tall peaked P waves, greater than 2.5 mm in amplitude in any lead, often best seen in lead II.

Left atrial enlargement is characterized by prolonged P wave duration, greater than 0.09–0.10 sec, and a negative terminal deflection in V1 >0.04 sec wide and >1 mm in depth. For *biatrial enlargement*, criteria for both right and left atrial enlargement are met.

Right ventricular hypertrophy (Table 2) is diagnosed when the height of the R wave in V1 or the depth of the S wave in V6 are greater than normal for age. Age-related criteria for normal QRS amplitude in leads V1 (which overlies the right ventricle) and V6 (which overlies the left ventricle) are summarized in Appendix 1. The normal neonate has right ventricular predominance, with gradual shifting to left ventricular dominance by 3–5 years of age. The T wave in V1 is normally upright at birth, inverts by 1 week of age, and may become upright once more when a mature ECG pattern is obtained, usually after 8 years of age. A persistently upright T wave in lead V1 after 1 week of age and before 8 years of age is indicative of right ventricular hypertrophy.

Left ventricular hypertrophy (Table 3) is diagnosed when the R wave amplitude in lead V6 or the S wave amplitude in lead V1 is greater than the 98th

Table 2: Right Ventricular Hypertrophy Criteria

R wave >98th percentile in lead V1
S wave >98th percentile in lead V6
R wave in V1 + S wave in V6 >98th percentile
R/S ratio >98th percentile in lead V1
Right axis deviation (>98th percentile of QRS in frontal plane)
qR pattern in V1
Upright T wave in V1 (1 week old to 8 years old)
RSR' pattern in lead V1, where R' >15 mm (<1 year old) or R' >10 mm (>1 year old)
Pure R wave in V1 >10 mm (newborn)
RVH (by voltage criteria) with strain pattern

Table 3: Left Ventricular Hypertrophy Criteria

R wave >98th percentile in lead V6
S wave >98th percentile in lead V1
R wave in V6 + S wave in V1 >98th percentile
Q wave >98th percentile in lead III or V6
R/S ratio >98th percentile in lead V6
LVH (by voltage criteria) with strain pattern

percentile for age. In the neonate, left ventricular forces in lead V6 range from 0 to 12 mm in height, with an average of 5 mm. The left ventricular voltage increases with age, and in general, an R wave greater than 27 mm in V6 at any age indicates left ventricular hypertrophy. Exceptionally deep S waves in V1 and V2, or very tall R waves in the inferior limb leads, are other indicators of left ventricular hypertrophy.

Biventricular hypertrophy is present when criteria for both right and left ventricular hypertrophy are present. The Katz–Wachtel criteria¹³ for biventricular hypertrophy is combined R + S wave voltage in the mid-precordial leads V3–V4 greater than 60 mm at any age.

Pacemakers: Pacemakers may be single or dual chamber systems, and are commonly described using the three-to-five-letter designation of the pacemaker nomenclature.¹⁴ The first letter states the chamber(s) paced; the second letter the chamber(s) sensed; the third letter designates the pacemaker’s response to intrinsic sensed beats; the fourth letter indicates programmable functions, including rate-responsiveness; the fifth letter indicates anti-tachycardia pacing functions. The more common designations are summarized in Table 4.

Table 4: Pacing Nomenclature and Common Programmable Generator Features

Feature	Measures and Options	Description
Pacing mode	<i>Single-chamber pacemakers</i> AAI, AOO, AAIR, VVI, VOO, and VVIR	Shorthand description of: chamber(s) paced (1st letter) and sensed (2nd letter), response to sensed beat (3rd letter), and whether pacemaker has a rate-adaptive sensor (R)
	<i>Dual-chamber pacemakers</i> DDD, DOO, VDD, DDI, DDDR, and DDIR	
Output	Voltage, current, and duration of pulse	Measures of energy generated by pacemaker when stimulus is delivered
Sensitivity	Atrial, ventricular, or both	Amplitude of intracardiac signal sensed as intrinsic atrial or ventricular activity
Rate	Lower rate limit, upper rate limit	Minimal intrinsic heart rate tolerated before pacing begins (lower rate limit); for dual-chamber pacemakers programmed to sense intrinsic atrial activity, maximal rate at which pacemaker tracks atrial activity in a 1 : 1 relation (upper rate limit)

A, atrium; D, dual; I, inhibited; O, none; R, rate-adaptive; V, ventricle

Indications for pacing are not reviewed in the text, but current recommendations are summarized below as a reference resource (Appendix 4).¹⁵ **Class I** indications are those for which there is evidence or general agreement that a given procedure or treatment is beneficial, useful, and effective. **Class II** indications are those for which there is conflicting evidence or a divergence of opinion about the usefulness or efficacy of a procedure or treatment. In **Class IIa** the weight of evidence or opinion is in favor of the procedure's usefulness or efficacy, while in **Class IIb** the usefulness or efficacy is less well established. **Class III** indications are those for which there is evidence or general agreement that a procedure or treatment is not useful or effective and in some cases may be harmful.

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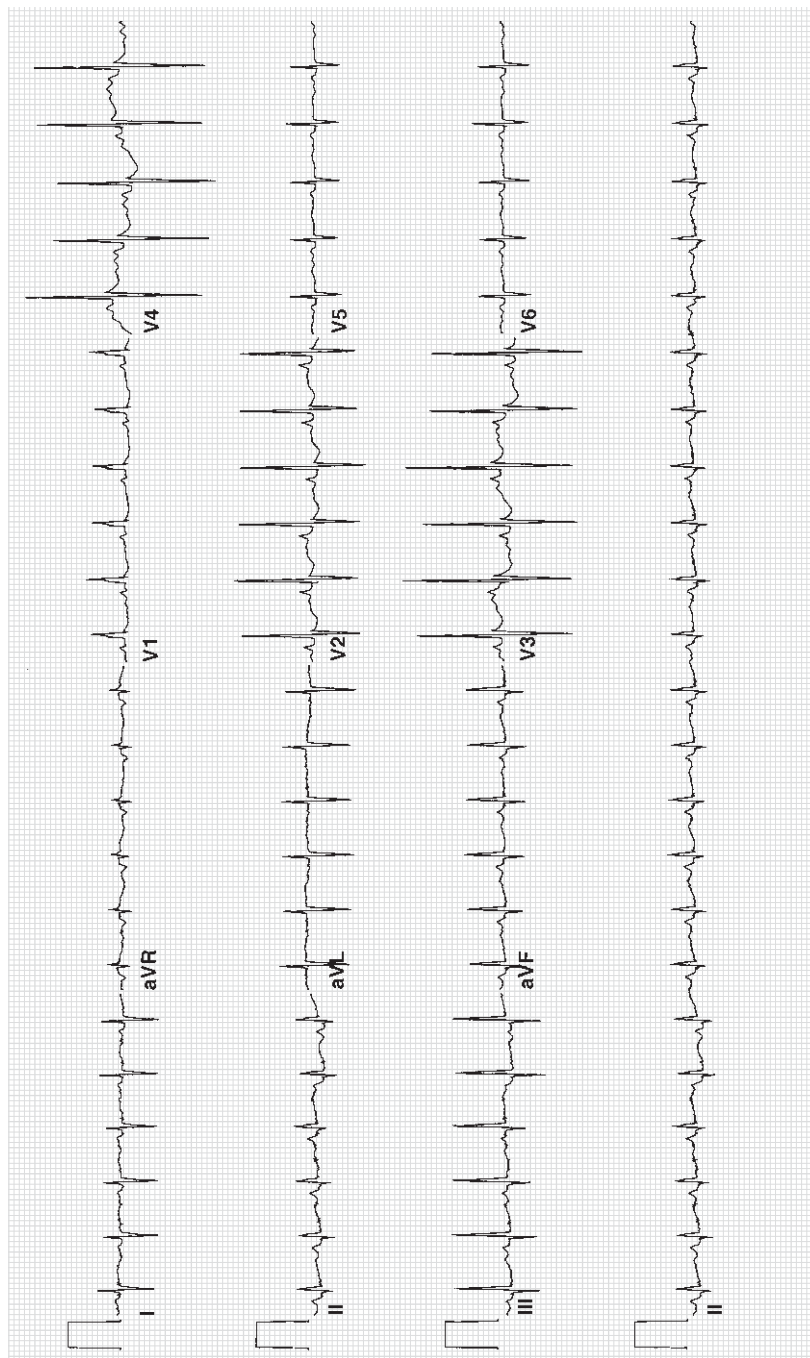


Figure 1 1-day-old infant.

Figure 1

- Normal sinus rhythm, 140 bpm
- QRS axis $+120^\circ$
- Pure R wave in V1
- S wave in V6 = 5 mm
- Nonspecific ST flattening

Normal ECG: 1 day old

- Heart rate: 94–155 bpm
- QRS axis: $+58^\circ$ to 192°
- Upright T waves in V1 are normal up to 7 days old
- Right ventricular predominance:
 - R wave in V1 ≤ 27 mm
 - R/S ratio in V1 = 0.2–9.8, mean 2.3
- R wave in V6: range 0–12 mm

Reference

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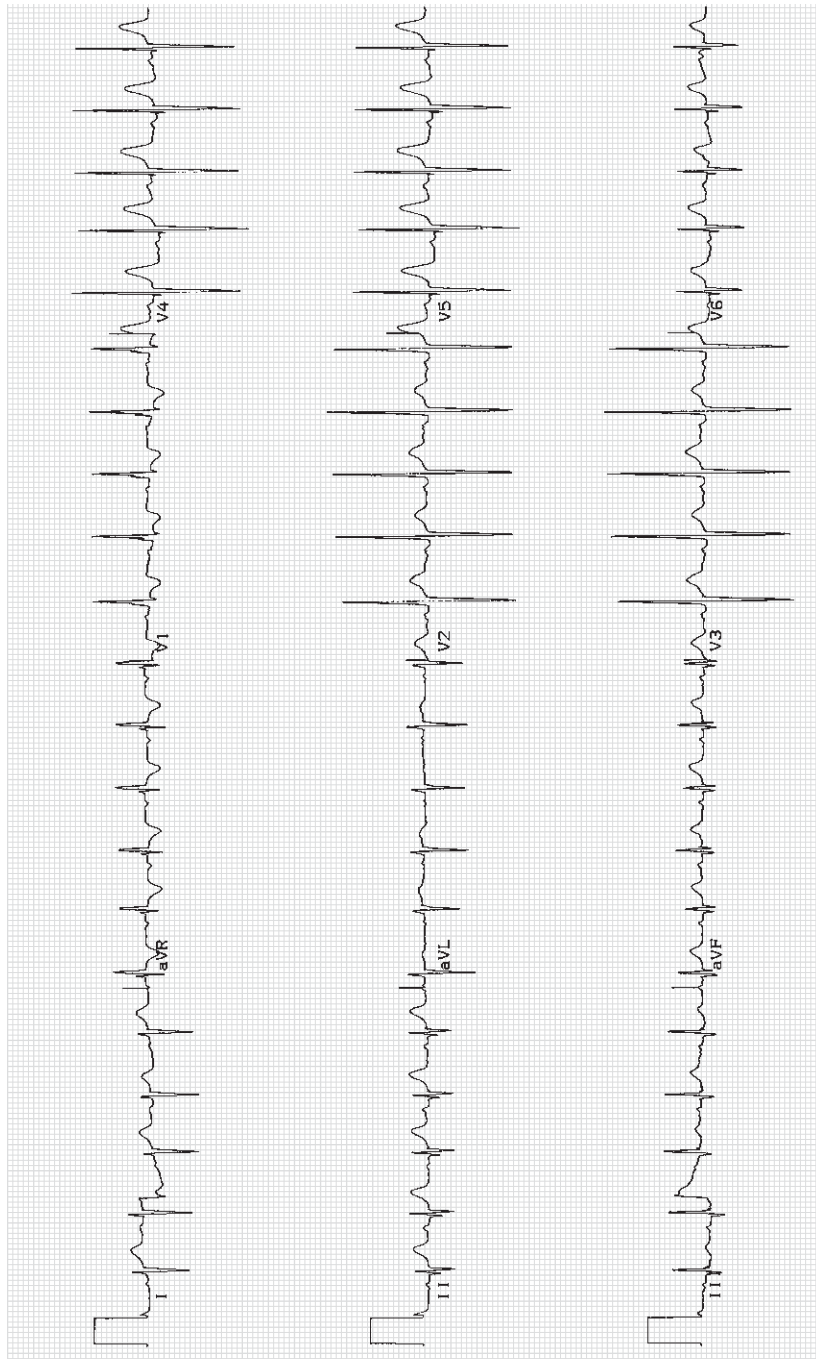


Figure 2 2-week-old infant.

Figure 2

- Normal sinus rhythm, 130 bpm
- QRS axis $+160^\circ$
- Tall R wave in V1
- S wave in V6 = 6 mm
- Inverted T waves in V1

Normal ECG: 2 weeks old

- Heart rate: 106–182 bpm
- QRS axis: $+65^\circ$ to $+160^\circ$
- R wave in V1 ≤ 22 mm
- R wave in V6 = 3–17 mm
- R/S ratio in V1 = 1.0–7.0

Reference

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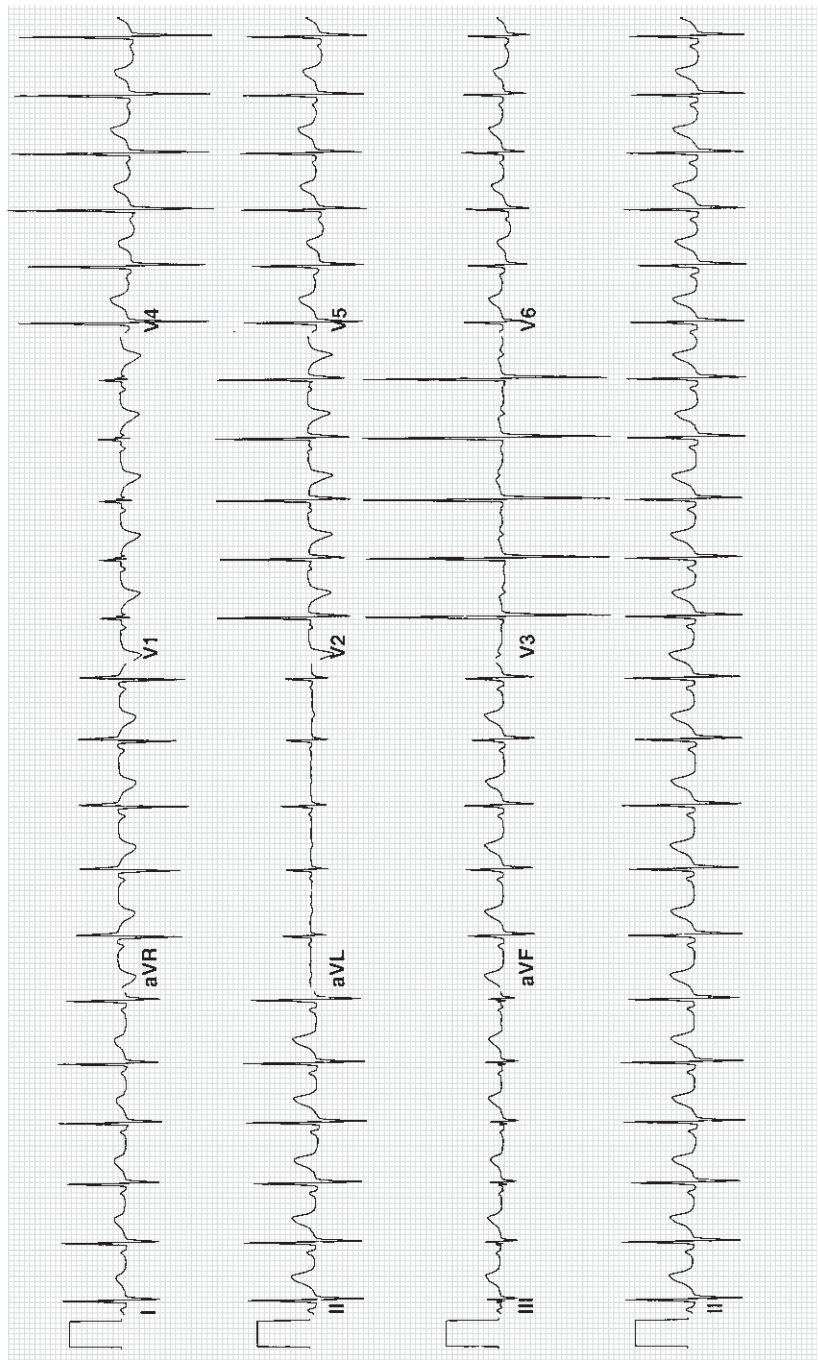


Figure 3 5-month-old female.

Figure 3

- Normal sinus rhythm, 130 bpm
- QRS axis $+30^\circ$
- Rsr' pattern in V1
- S wave in V6 = 3 mm

Normal ECG: 5 months old

- Heart rate: 105–185 bpm
- QRS axis: $+7^\circ$ to $+105^\circ$
- R wave in V1 ≤ 20 mm
- R wave in V6 = 6–23 mm
- R/S ratio in V1 = 0.2–6.0
- Q in lead III ≤ 7 mm

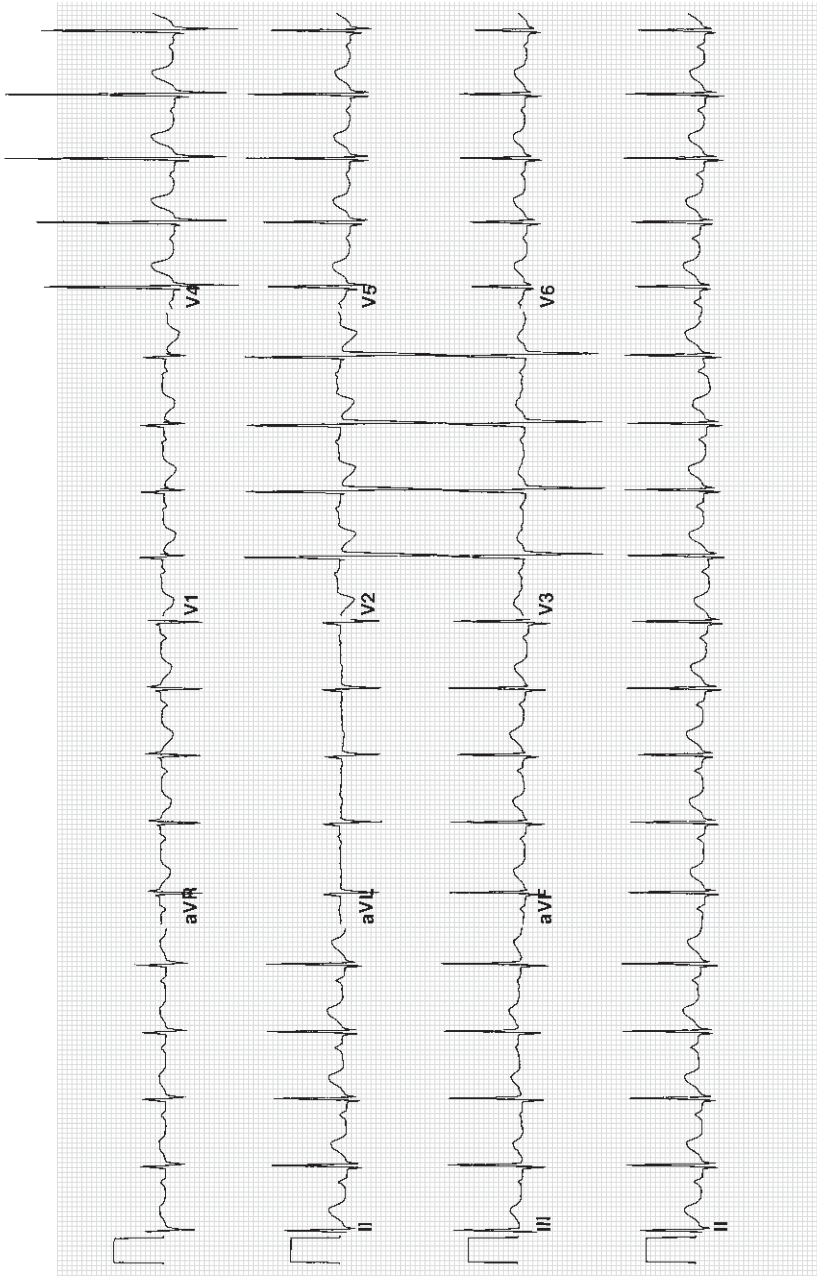


Figure 4 Asymptomatic 1-year-old male.

Figure 4

- Normal sinus rhythm, 110 bpm
- QRS axis $+80^\circ$
- Small q waves in inferior and lateral leads
- rS in V1 progressing to dominant R wave in V6

Normal ECG: 1 year old

- Heart rate: 90–150 bpm
- QRS axis: $+8^\circ$ to $+100^\circ$
- R wave in V1: 3–18 mm
- R wave in V6: 6–23 mm
- R/S ratio in V1: 0.1–4.2
- Q wave in lead III ≤ 5 mm

Reference

Walsh EP, Lang P, Ellison RC, Zierler S, Harned HS, Miettinen OS. Electrocardiogram of the premature infant at 1 year of age. *Pediatrics* 1986;77:353–356.

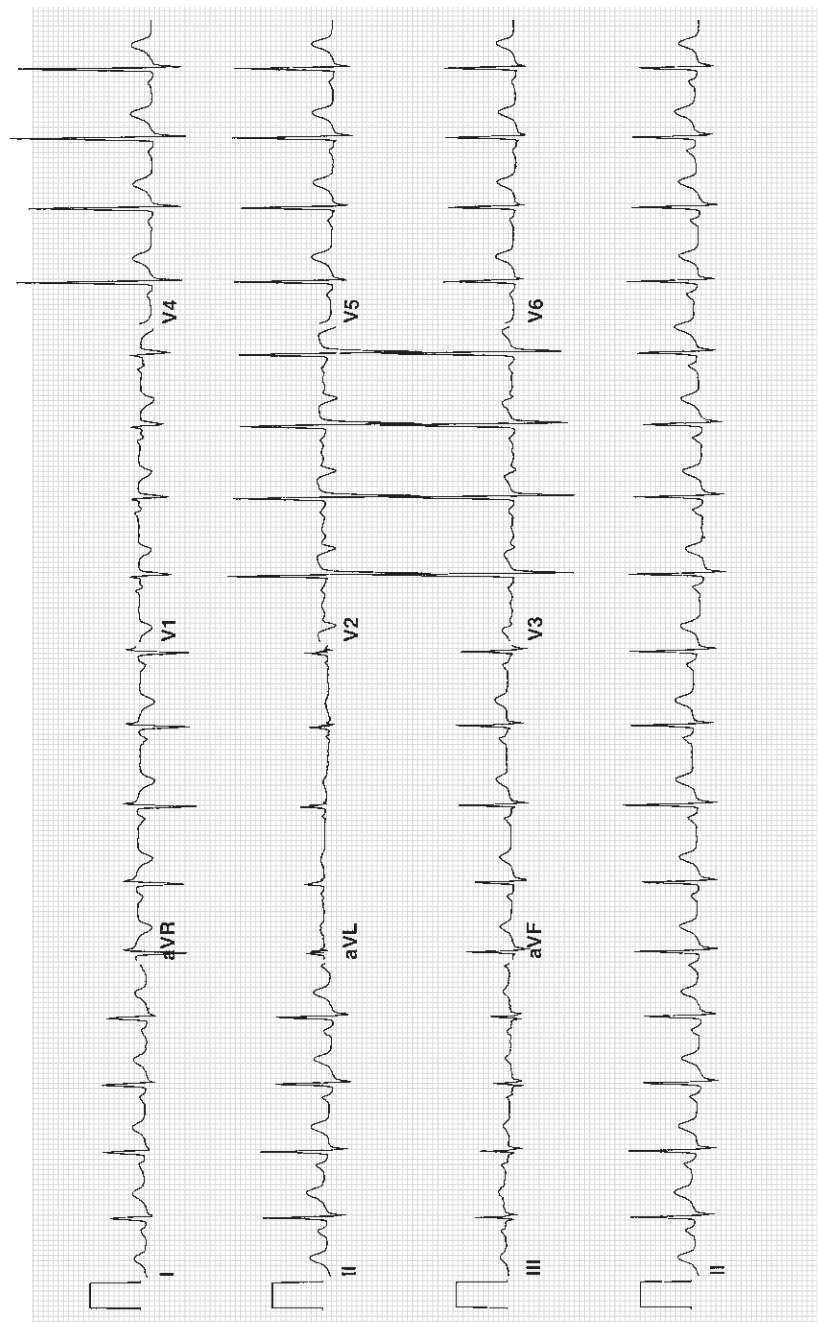


Figure 5 Asymptomatic 5-year-old female.

Figure 5

- Normal sinus rhythm, 105 bpm
- QRS axis $+50^\circ$
- rS in V1, progressing to dominant R wave in V6
- Normal precordial voltage

Normal ECG: 5 years old

- Heart rate: 70–140 bpm
- QRS axis: $+7^\circ$ to $+105^\circ$
- R wave in V1: 2–18 mm
- R wave in V6: 9–25 mm
- R/S ratio in V1: 0–2.8

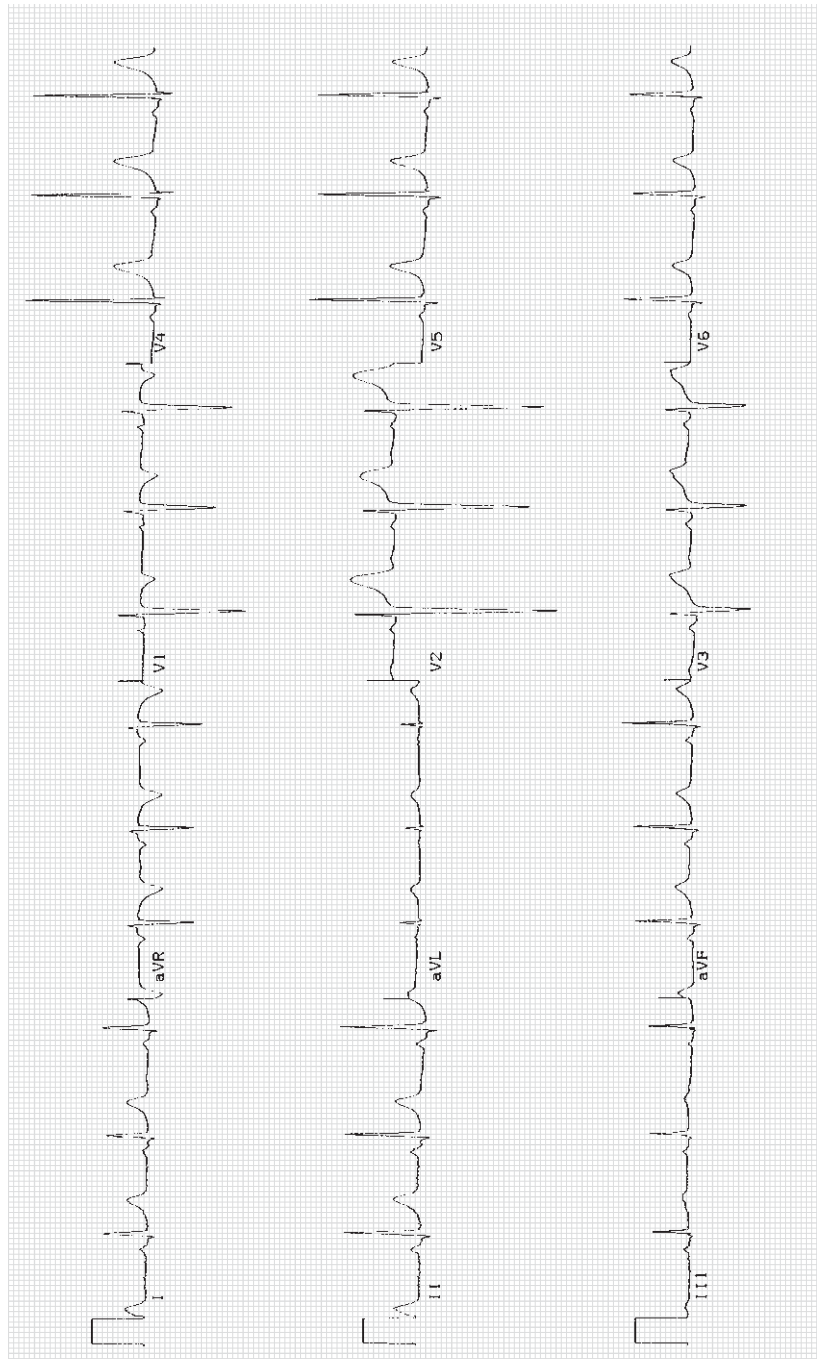


Figure 6 Asymptomatic 8-year-old female.

Figure 6

- Normal sinus rhythm, 75 bpm
- QRS axis: $+60^\circ$
- rS in V1, qR in V6
- Normal precordial voltage

Normal ECG: 8 years old

- Heart rate: 60–130 bpm
- QRS axis: $+9^\circ$ to 115°
- R wave in V1: 0.5–10 mm
- R wave in V6: 10–26 mm
- R/S ratio in V1: 0–1.9
- Q wave in lead III ≤ 3 mm
- Transition from negative to positive T wave in right precordial leads

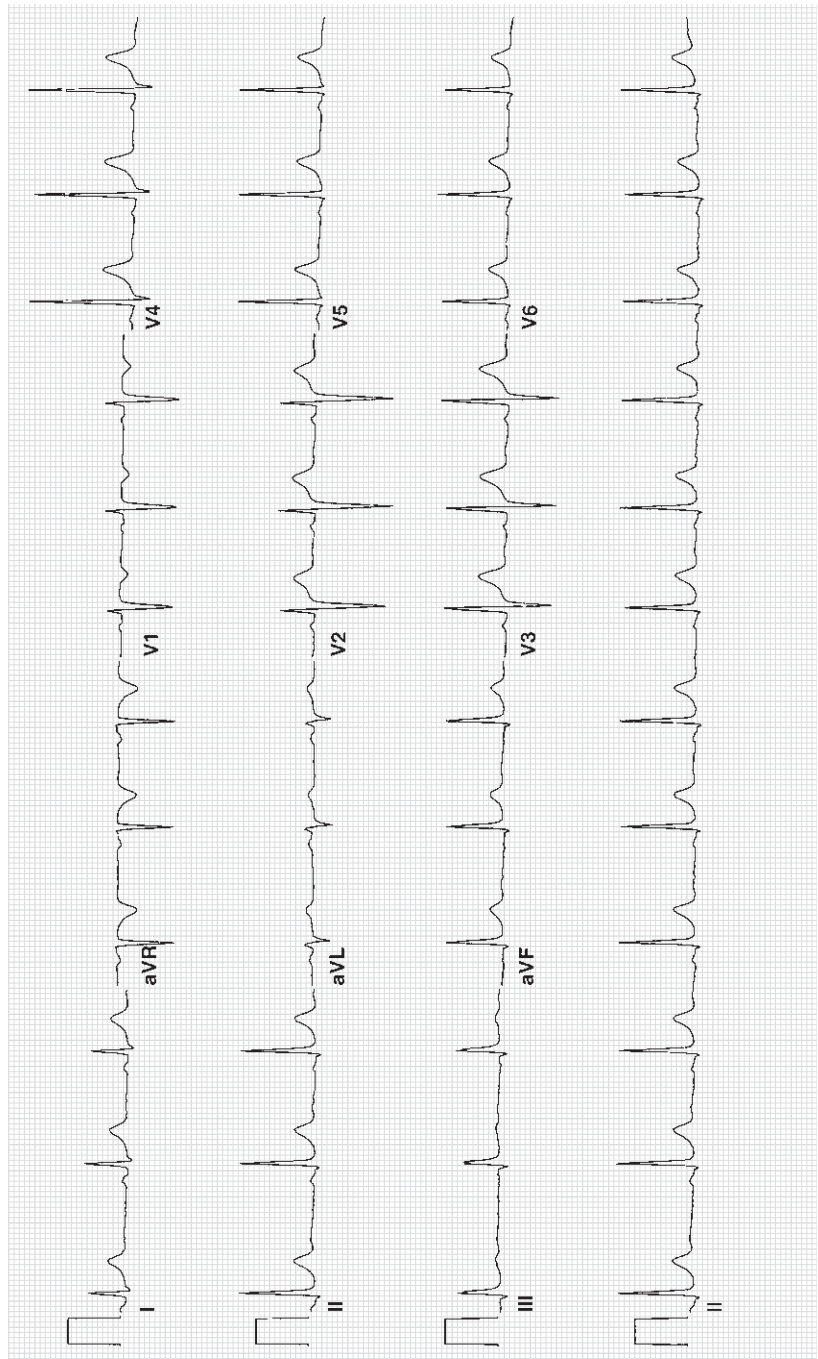


Figure 7 Healthy 15-year-old male.

Figure 7

- Normal sinus rhythm, 70bpm
- QRS axis: $+60^\circ$
- rS in V1 progressing to qR in V6
- Normal precordial voltage

Normal ECG: 15 years old

- Heart rate: 66–120bpm
- QRS axis: $+11^\circ$ to $+13^\circ$
- R wave in V1: 0.5–10mm
- R wave in V6: 7–23mm
- R/S ratio in V1: 0–1.8

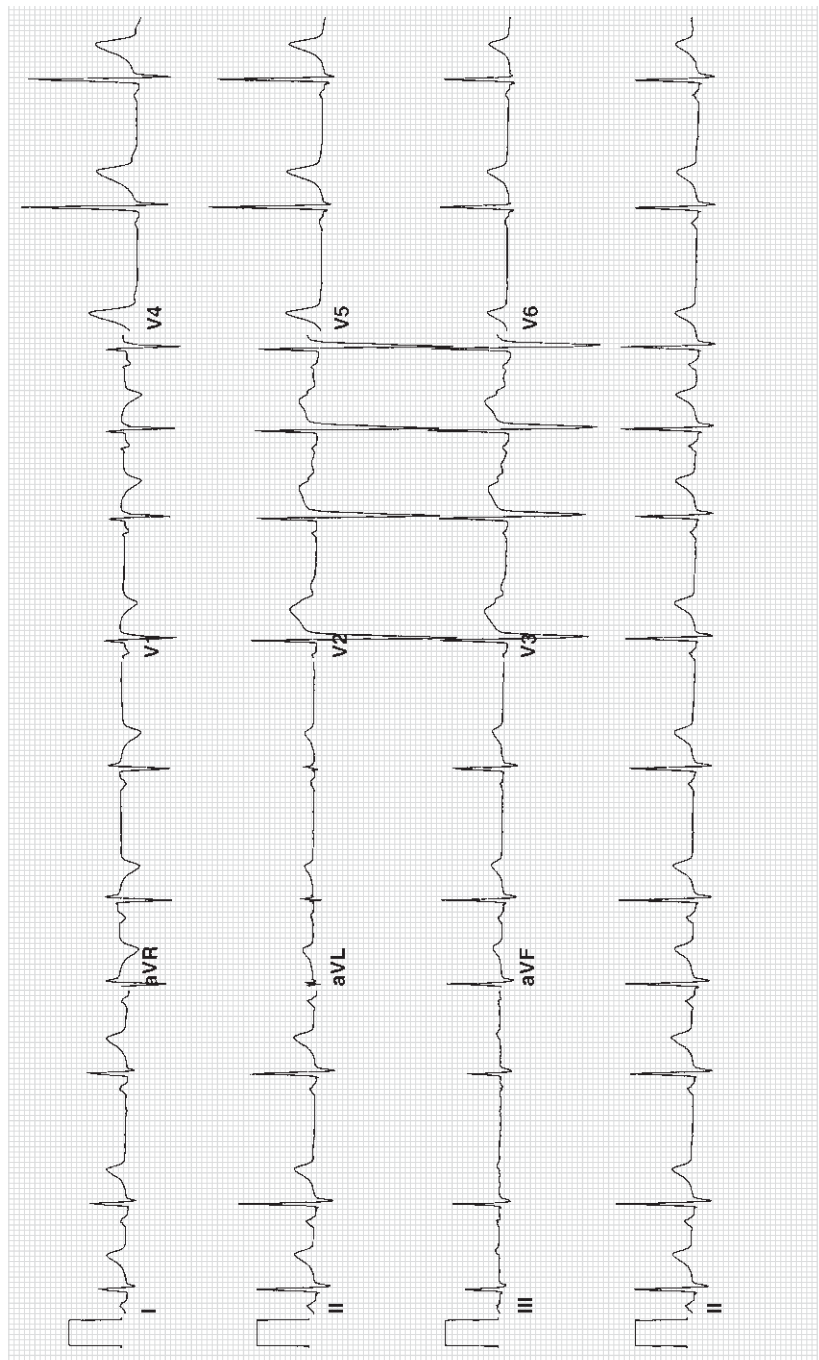


Figure 8 8-year-old boy referred for irregular heart rhythm.

Figure 8

- Sinus rhythm with phasic variation in heart rate
- Normal P wave precedes each QRS complex
- Normal PR interval

Sinus arrhythmia

- Normal P wave morphology with variable P–P interval
- Respiratory form of sinus arrhythmia: sinus rate gradually increases during inspiration, slows with expiration
- Very common in young children, ages 2–10 years
- Normal variant, associated with increased vagal tone

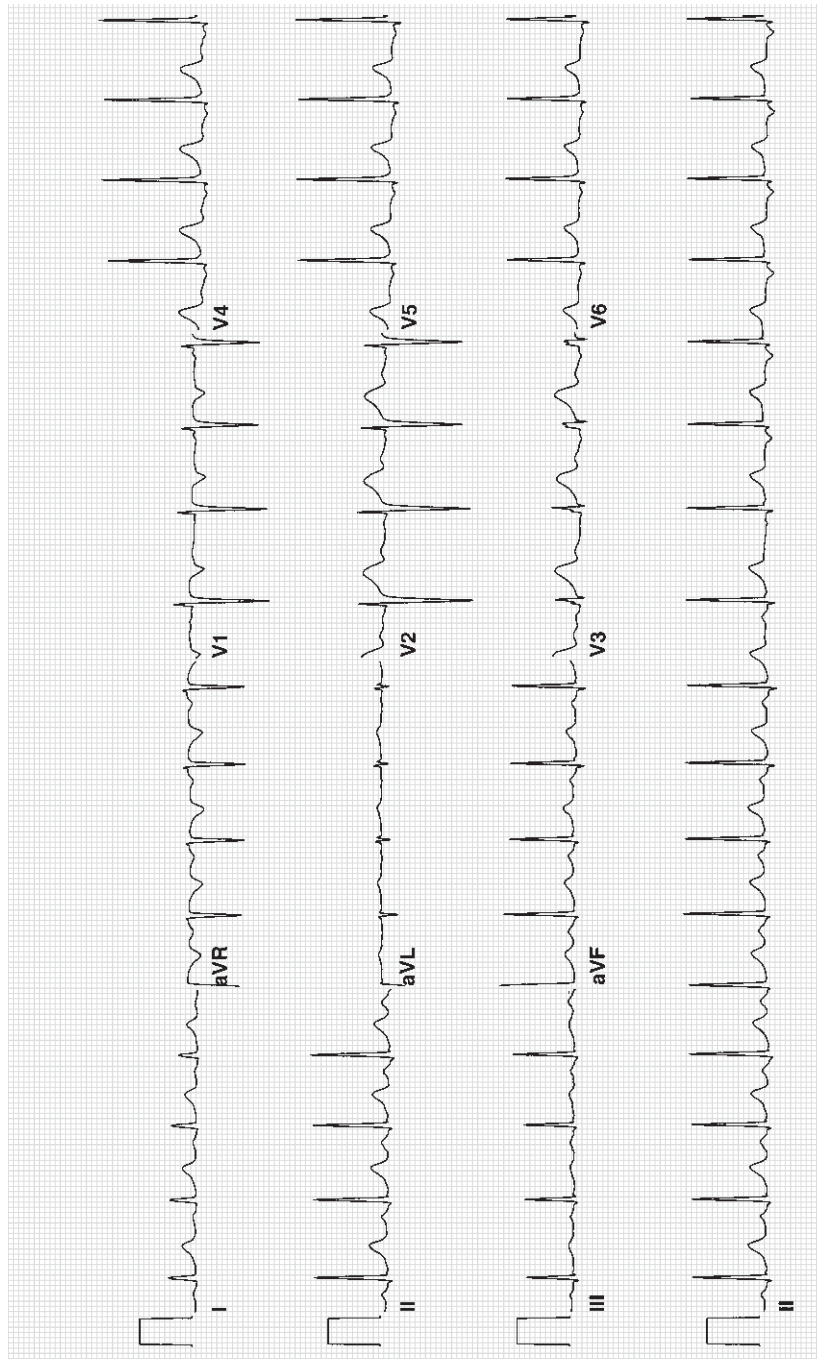


Figure 9 Asymptomatic 14-year-old female.

Figure 9

- Heart rate 100 bpm
- P wave morphology shifts gradually
- P wave is initially positive in I, II, III, aVF, consistent with sinus rhythm
- As sinus rate slows, P wave changes to become negative in lead II (see bottom rhythm strip) and V6, suggesting a left atrial rhythm
- QTc interval at upper limits of normal (0.45 sec)

Wandering atrial pacemaker

- Shifting location of atrial pacemaker
- P wave morphology, amplitude, and axis shifts gradually
- Not associated with premature beats or tachycardia

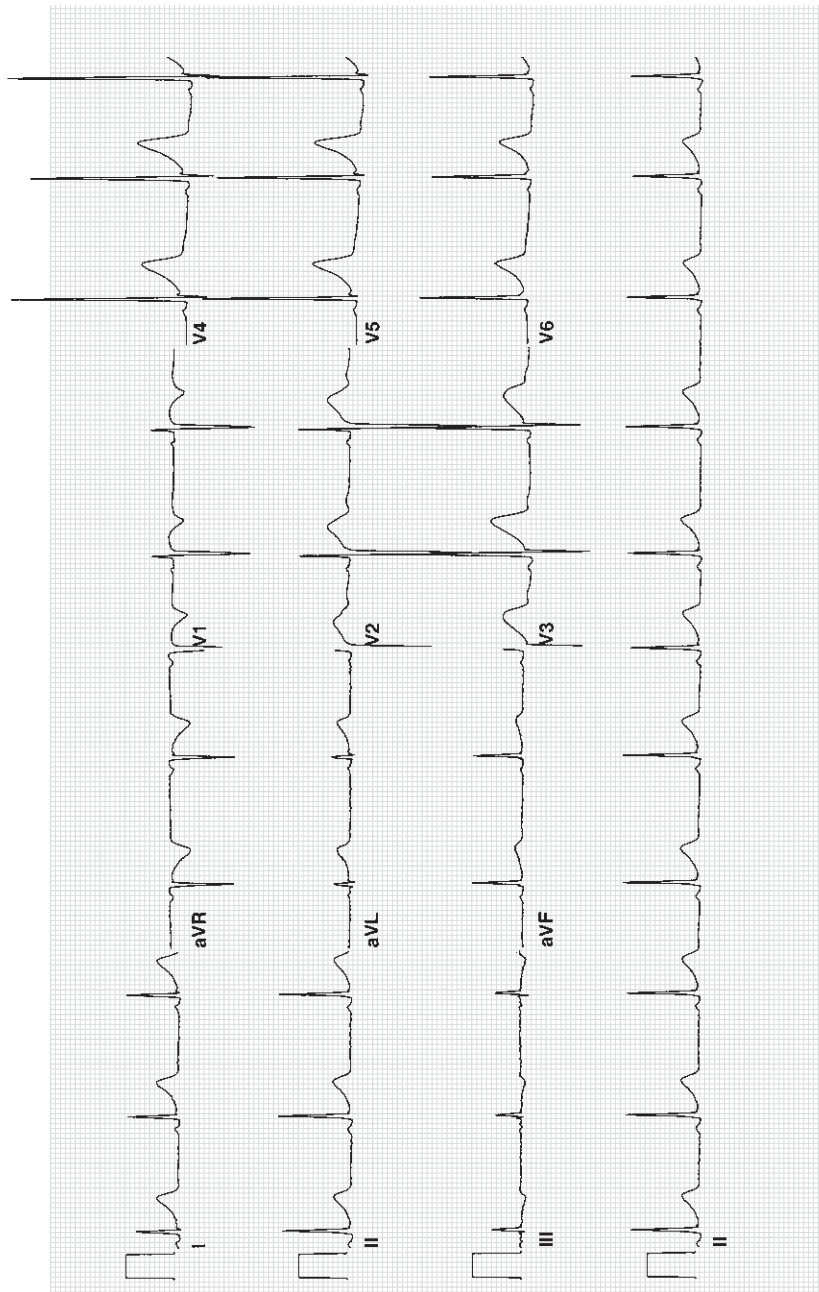


Figure 10 9-year-old male.

Figure 10

- Sinus rhythm, 60 bpm
- Tall T waves in left precordial leads
- Mild ST segment elevation noted in leads I, II, and across precordium

Early repolarization

- Elevated ST segment by 1–4 mm, especially seen in leads V2–V6
- ST segment shows upward concavity
- ST segment elevation <25% of T wave height
- Downstroke of R wave may show slur or notch
- Large, symmetric T waves
- Normal variant, common in adolescents

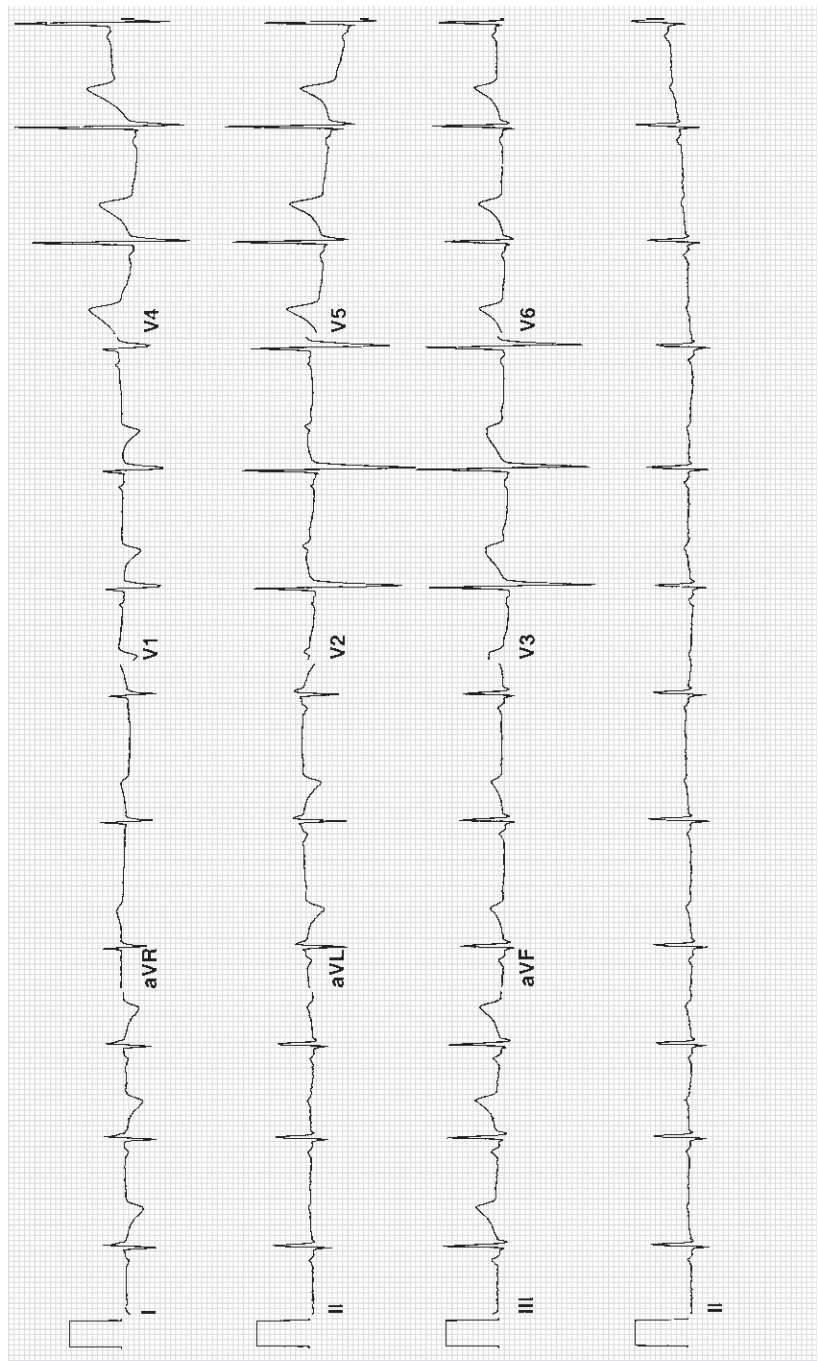


Figure 11 9-year-old boy referred for murmur evaluation.

Figure 11

- Heart rate 70 bpm
- Negative P wave in leads I, aVL with positive P waves in V3–V6
- Deep Q waves in I, aVL; normal Q waves in V5, V6
- rSR' in leads I, aVL with normal QRS in V5–V6
- Negative T waves in I, aVL; normal precordial T waves

Limb lead reversal artifact

- Most common lead reversal artifact is due to exchanged positions of right and left arm electrodes.
- Normal P, QRS, and T complexes in precordial leads, in contrast to inverted complexes in limb leads I, aVL

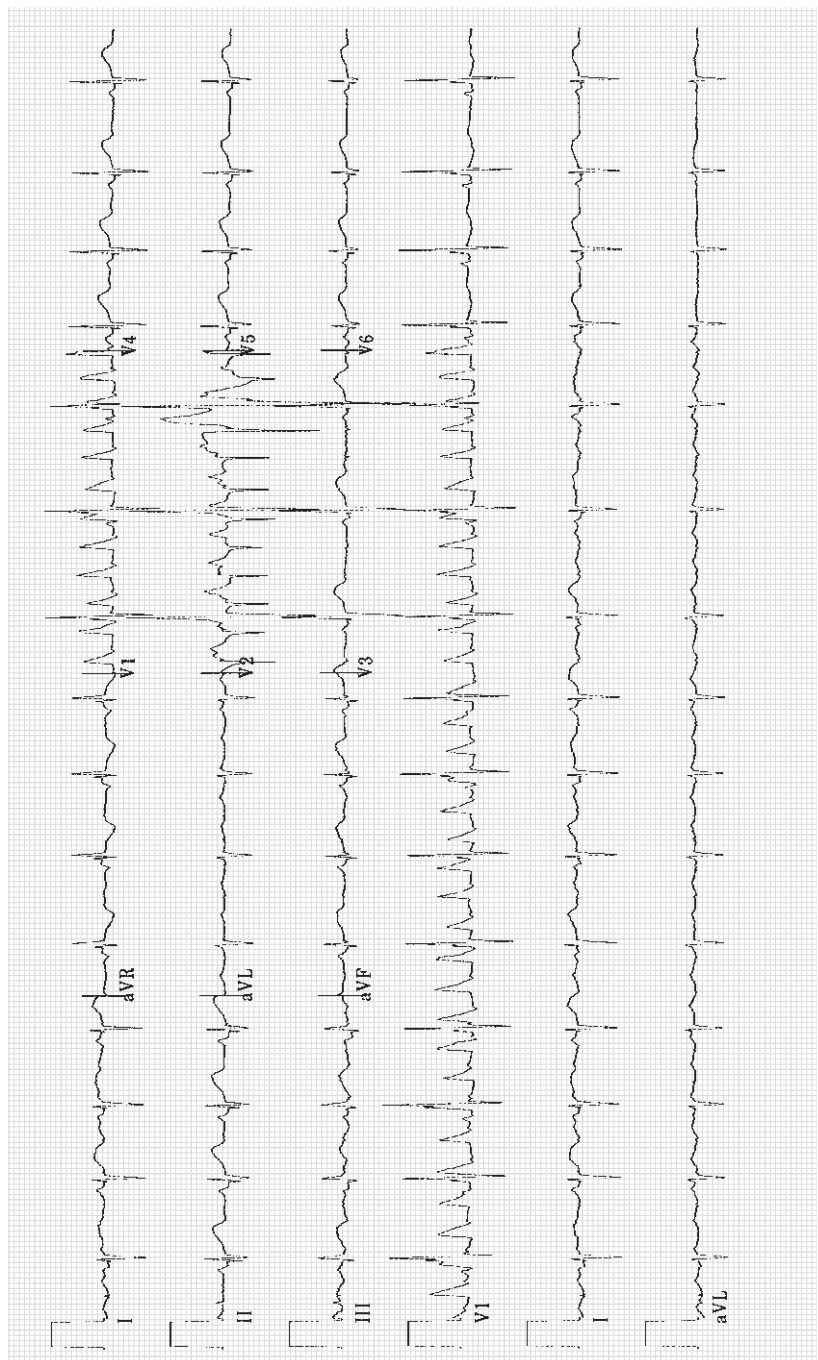


Figure 12 5-day-old baby being calmed during ECG acquisition.

Figure 12

- Sinus P waves are present, with giant “P waves” superimposed

Movement artifact

- Rhythmic rapid artifact, mimics atrial, or ventricular tachycardia.
- Often present with hiccups, or “patting the baby” on the back

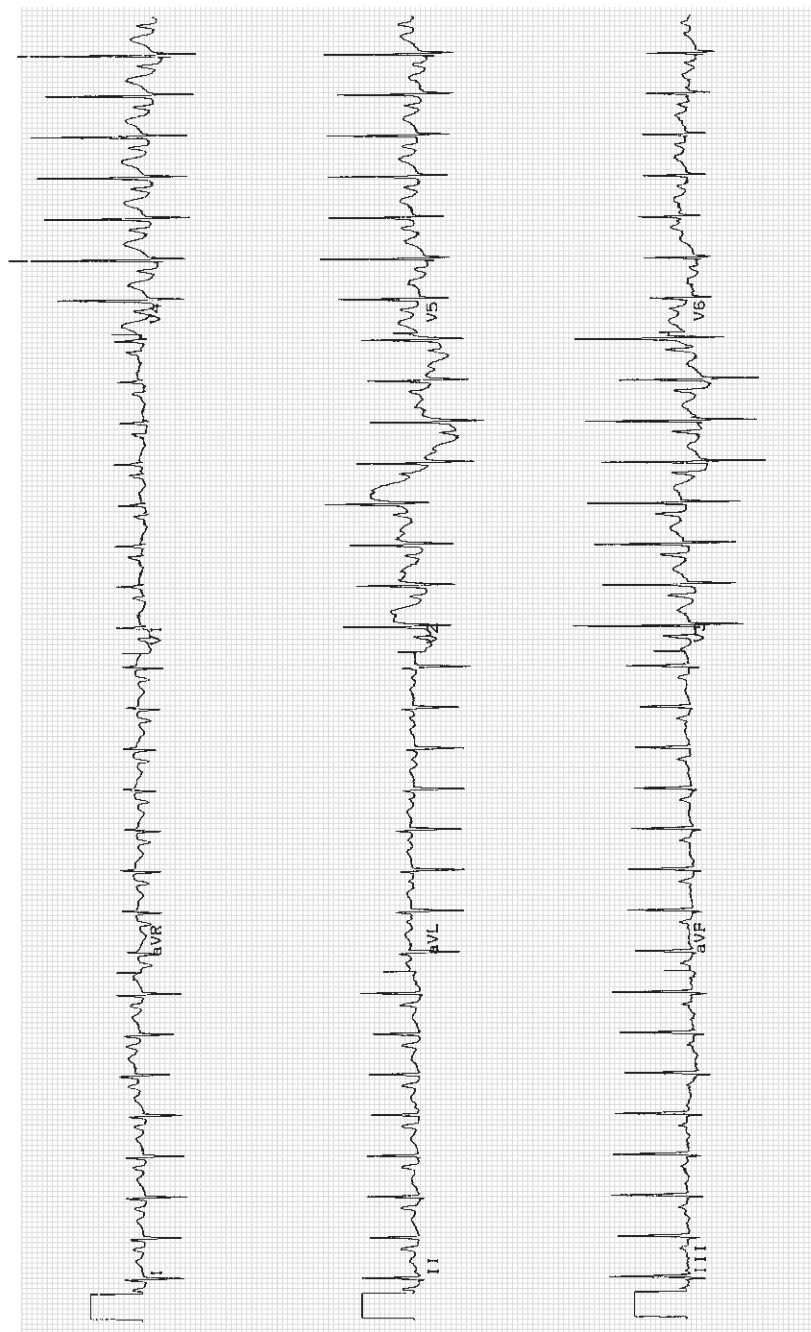


Figure 13 3-week-old infant with chronic lung disease.

Figure 13

- Sinus tachycardia, 187bpm
- QRS axis $+110^\circ$
- Tall, peaked P waves in leads II, V1–V5

Right atrial enlargement

- Tall, peaked P waves >2.5 mm in any lead, best seen in leads II, III, aVF, V1, V2
- Normal newborns may have P wave amplitude up to 3 mm in first few days of life

Reference

Davignon A, *et al.* Percentile charts: ECG standards for children. *Pediatr Cardiol* 1980;1:133–152.

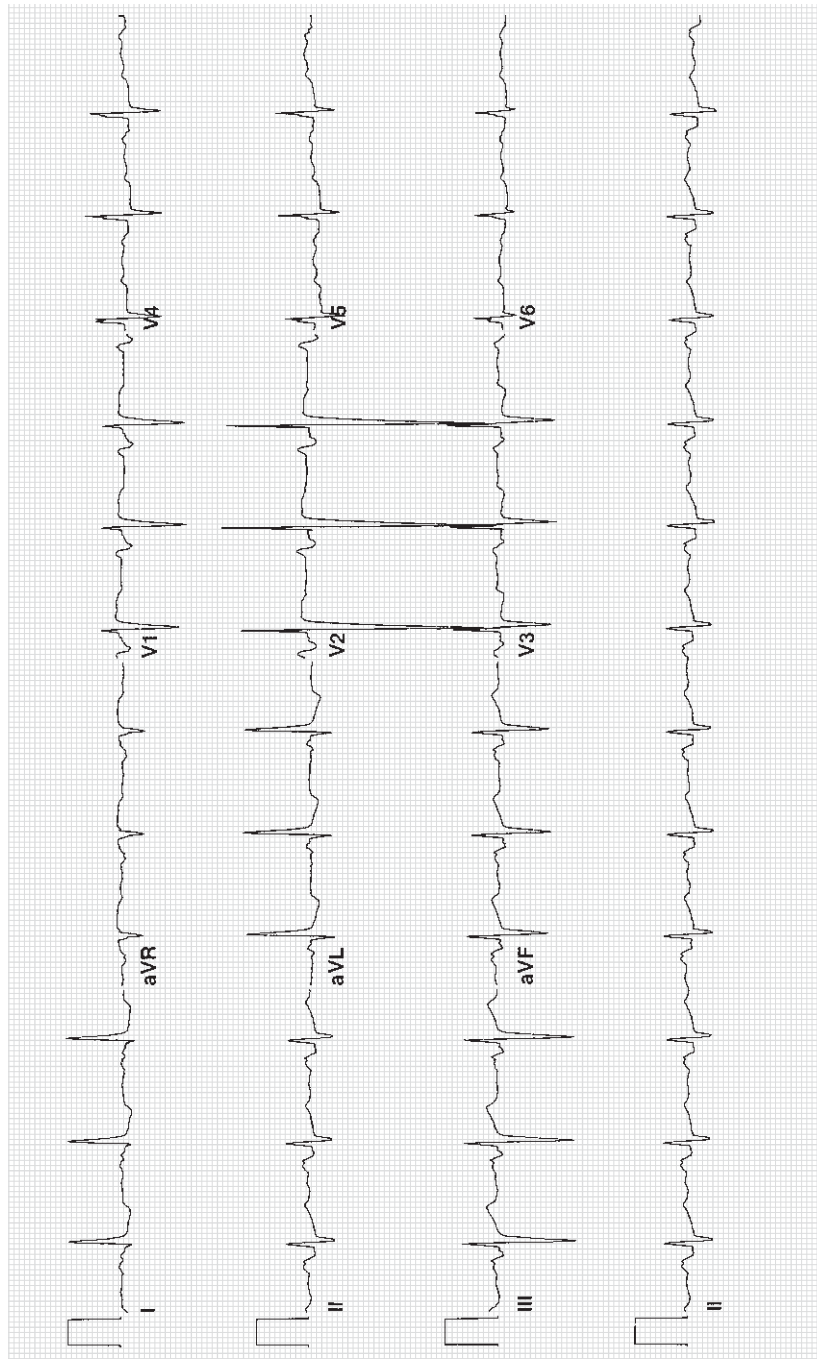


Figure 14 16-year-old girl with tricuspid atresia status post Fontan procedure.

Figure 14

- Sinus rhythm, 76 bpm
- QRS axis -20° ; left axis deviation
- PR interval 0.210 sec: first degree atrioventricular block
- Broad notched P waves in leads I, II, aVF
- Wide, deep negative component of P waves in V1
- Deep S wave in V2 suggests left ventricular hypertrophy
- Nonspecific ST wave changes

Left atrial enlargement

- Biphasic P wave in lead V1 with deep terminal negative deflection: $>0.04 \times 1.0$ mm
- Broad notched P wave, with duration >0.10 – 0.12 sec
- Note: A biphasic P wave in V1 of normal duration without deep terminal negative component is common in normal children

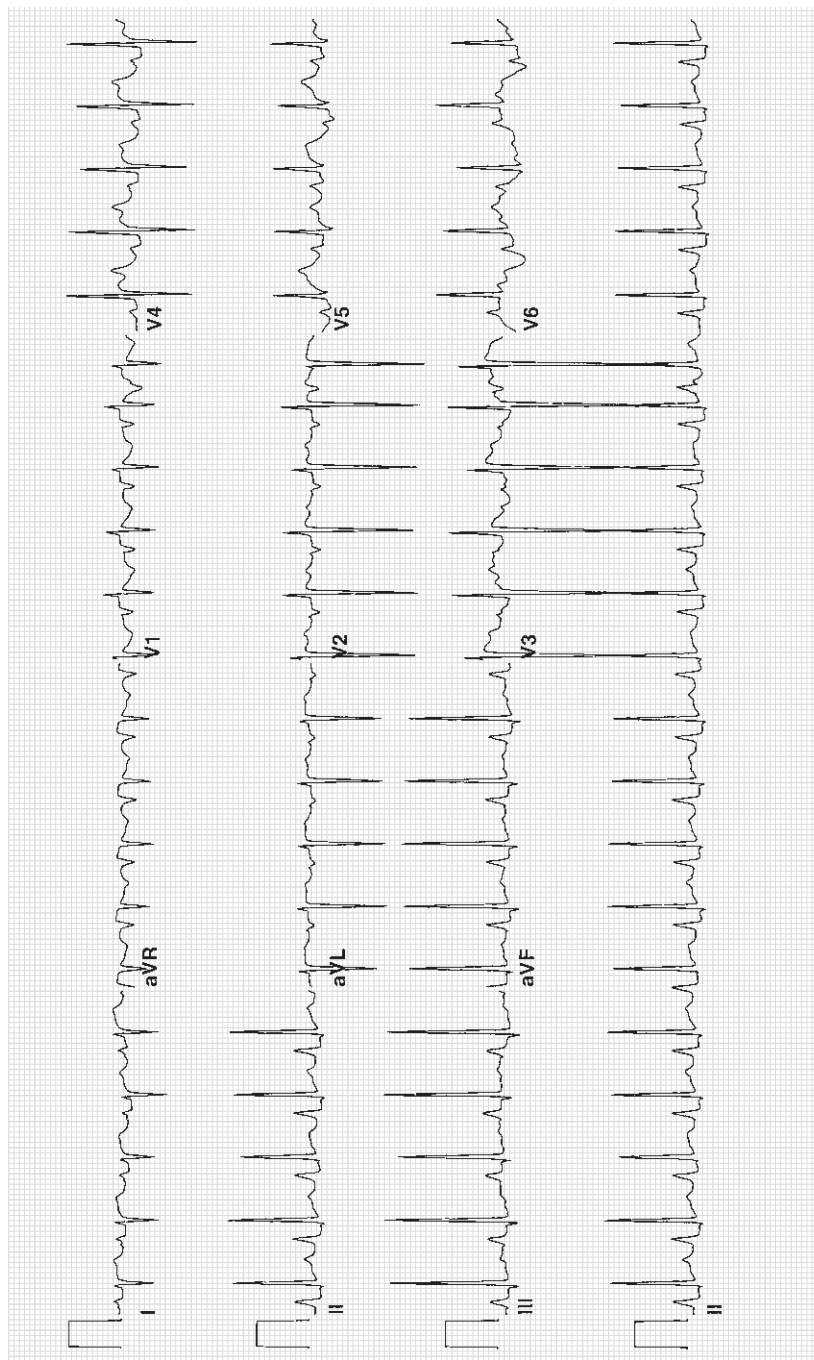


Figure 15 4-week-old infant with transposition of the great arteries, ventricular septal defect, status post-arterial switch operation.

Figure 15

- Normal sinus rhythm, 125 bpm, isolated PAC
- Tall peaked P waves in leads II, III, aVF
- Deep negative terminal component of P wave in V1
- Tall R waves in leads II, III, aVF, deep S wave in V2 suggest left ventricular predominance
- Nonspecific ST wave changes

Biatrial enlargement

- Increased P wave amplitude (>2.5 mm) and duration (>0.10 – 0.12 sec) in limb leads
- Large biphasic P wave in V1:
 - Initial positive component >1.5 mm
 - Deep terminal negative component (>0.04 sec $\times$ 1.0 mm)

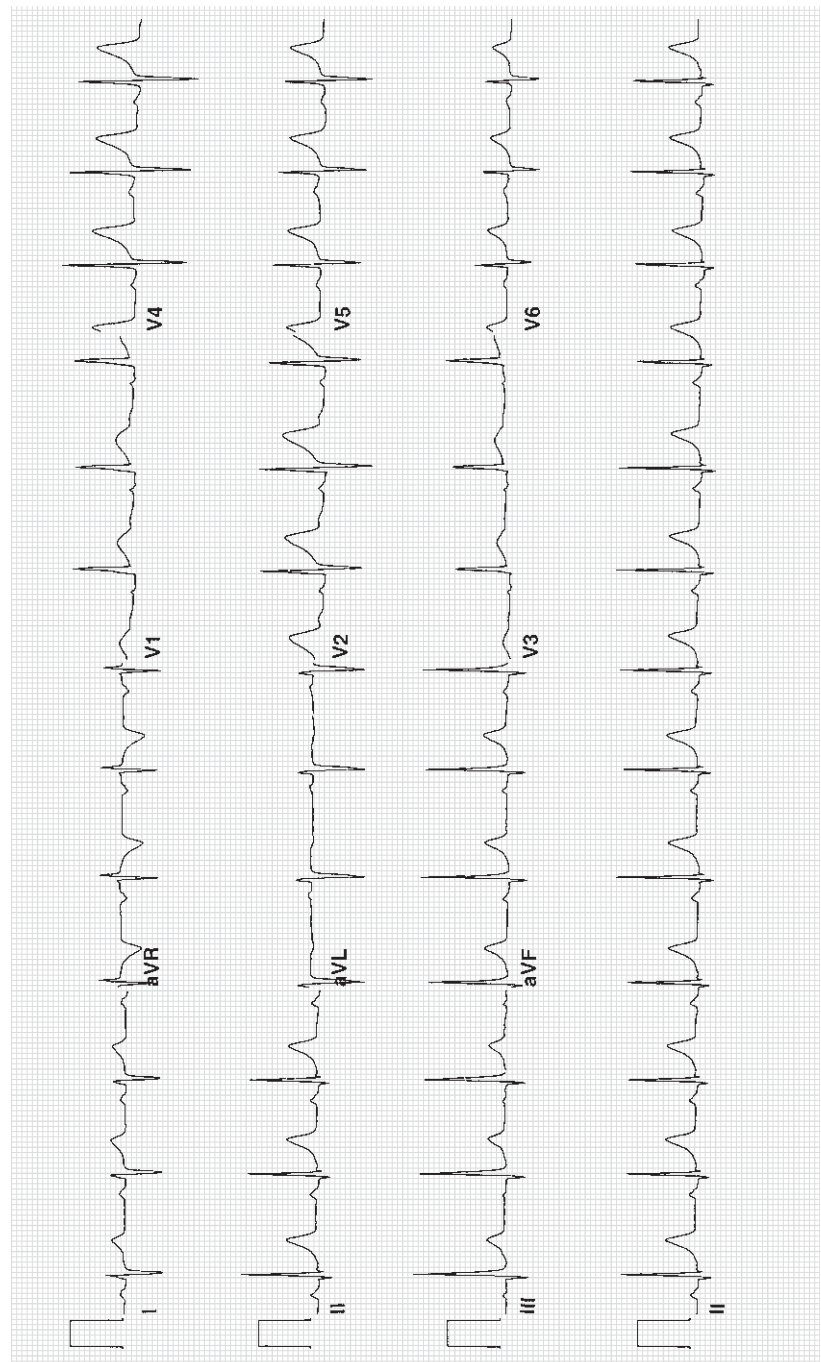


Figure 16 9-year-old boy with loud systolic murmur at left upper sternal border.

Figure 16

- Normal sinus rhythm, 80bpm
- QRS axis: 102°
- Pure R wave in V1
- S wave in V6 = 6mm
- Upright T wave in V1

Right ventricular hypertrophy: 9 years old

- R wave in V1 >13 mm
- S wave in V6 >4 mm
- R/S ratio in V1 >2
- Q wave or qR pattern in right precordial leads
- Upright T wave in V1 after first week of life, and before adolescent ECG pattern
- Right axis deviation $>+115^\circ$

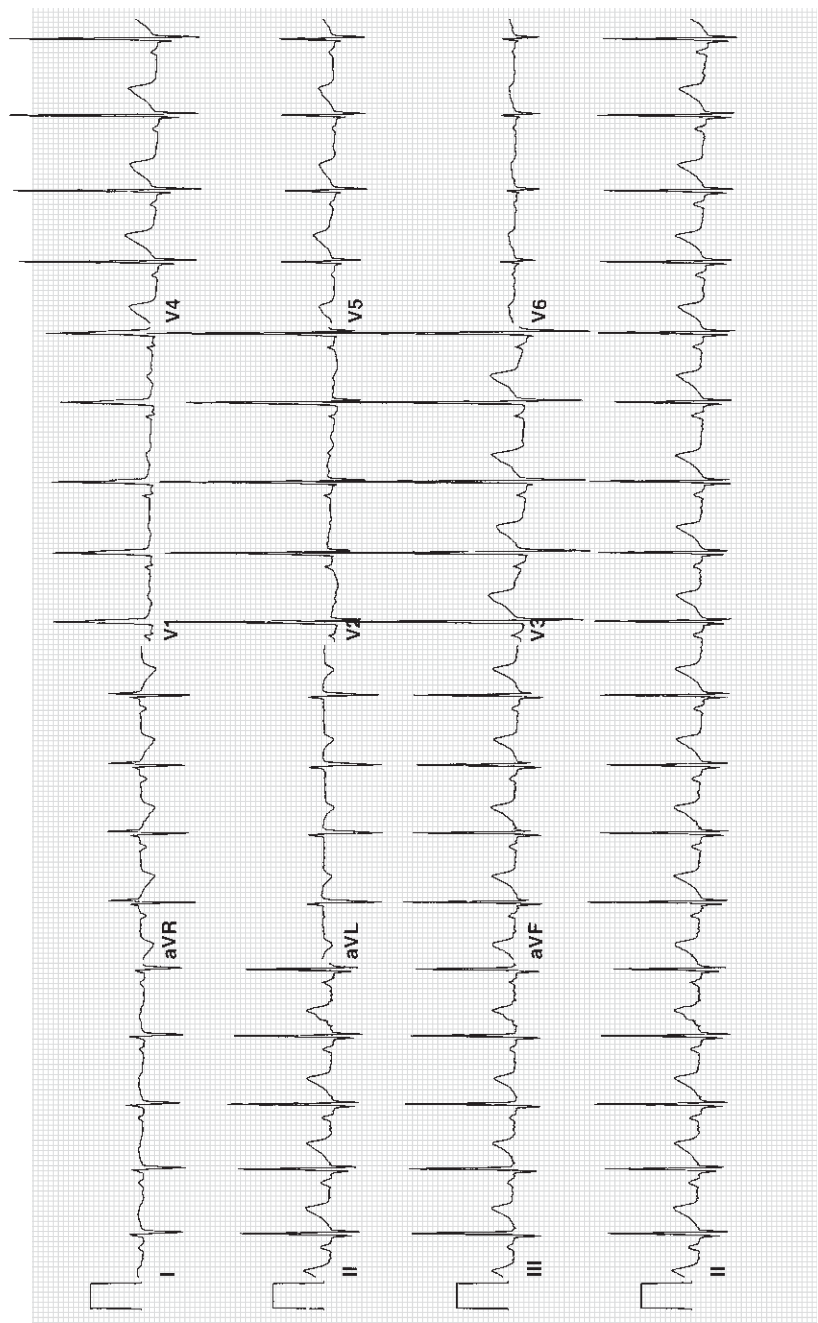


Figure 17 7-month-old infant with loud systolic murmur at left upper sternal border.

Figure 17

- Normal sinus rhythm, 110 bpm
- QRS axis $+110^\circ$; right axis deviation
- qR pattern in V1
- Tall R wave in V1 = 21 mm
- Upright T wave in V1

Right ventricular hypertrophy: 7 months old

- R wave in V1 >20 mm (in absence of right bundle branch block)
- S wave in V6 >8 mm
- Q wave or qR pattern in V1
- Right axis deviation: QRS axis $>105^\circ$
- Upright T wave in V1 after first week of life

* For severe right ventricular hypertrophy, see pulmonary stenosis, p. 115.

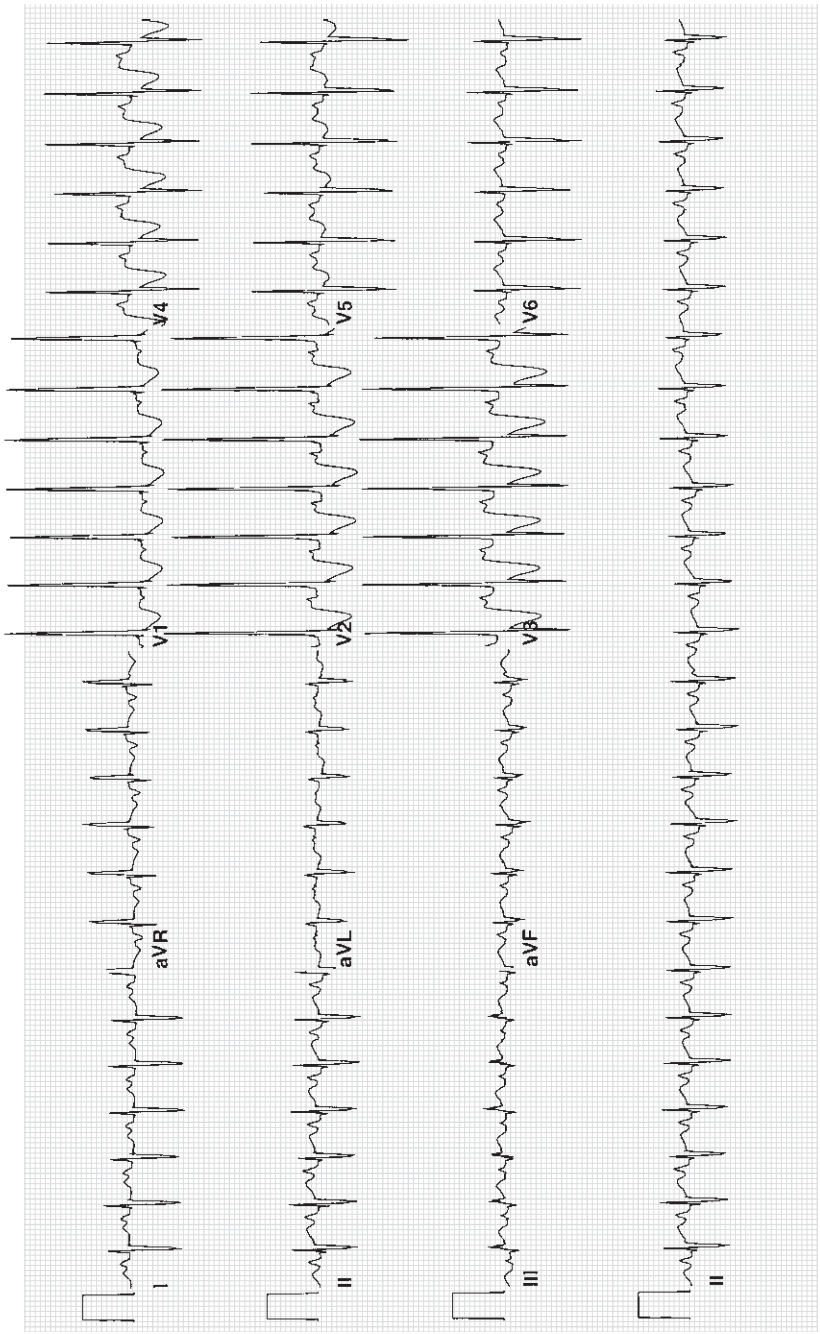


Figure 18 1-week-old infant with harsh systolic murmur.

Figure 18

- Normal sinus rhythm, 160 bpm
- QRS axis $+180^\circ$
- rsR' in lead V1, R' = 26 mm
- S wave in V6 = 13 mm
- ST segment depression in V1–V5
- QRS–T wave discordance

Right ventricular hypertrophy with strain: neonate

- R wave in V1 >27 mm
- rsR' in V1, R' >15 mm
- S wave in V6 >10 mm
- qR pattern in V1
- Right axis deviation $>200^\circ$
- ST segment depression in right precordial leads
- Wide QRS–T angle $>90^\circ$

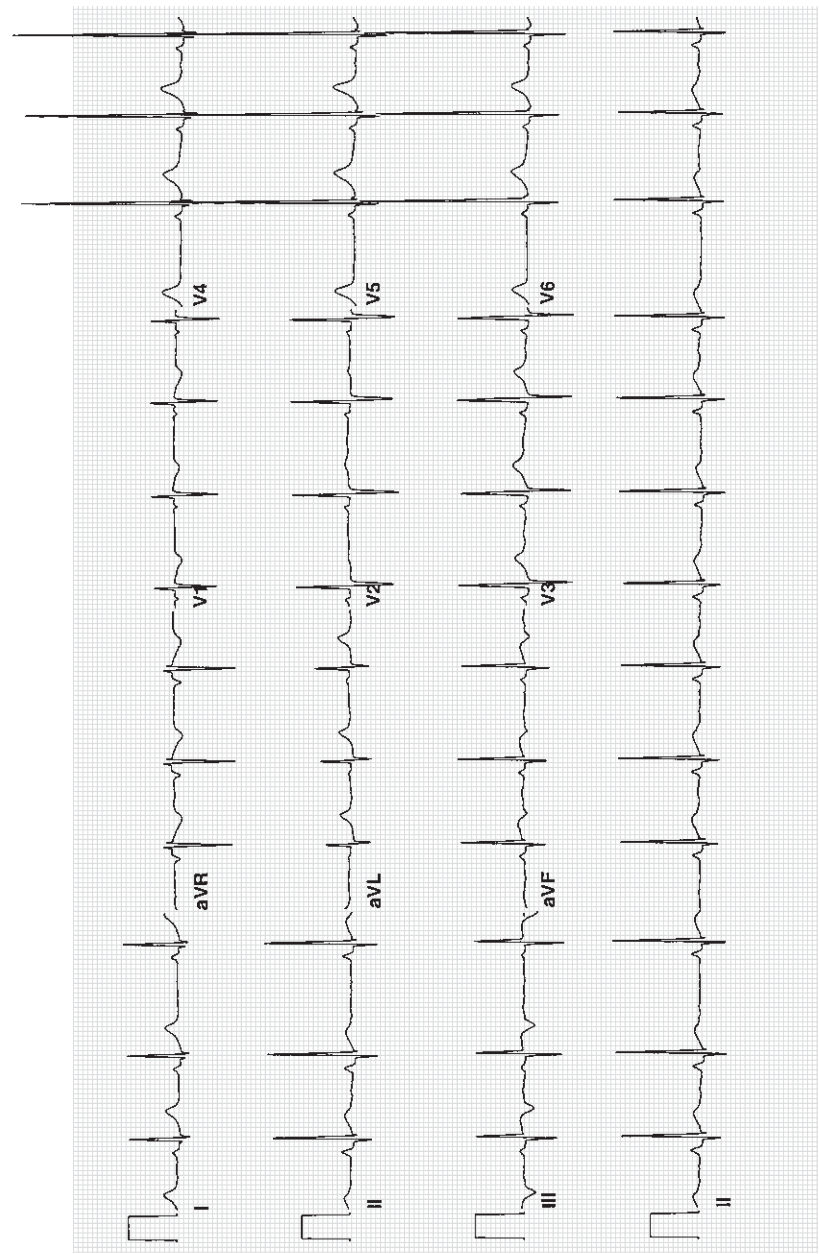


Figure 19 5-year-old girl with systolic murmur at right upper sternal border.

Figure 19

- Sinus rhythm with sinus arrhythmia, 80 bpm
 - QRS axis $+45^\circ$
 - R wave in V6 >30 mm
 - Q wave in lead III = 8 mm, lead V6 = 6 mm
- Left ventricular hypertrophy
- Early repolarization changes

Left ventricular hypertrophy: 5 years old

- R wave in V6 >25 mm
- Amplitude of R wave in V5 or V6 plus S wave in V1 >47 mm
- S wave in V1 ≥ 25 mm
- Deep Q waves in lead III (≥ 4 mm) or V6 (≥ 5 mm)

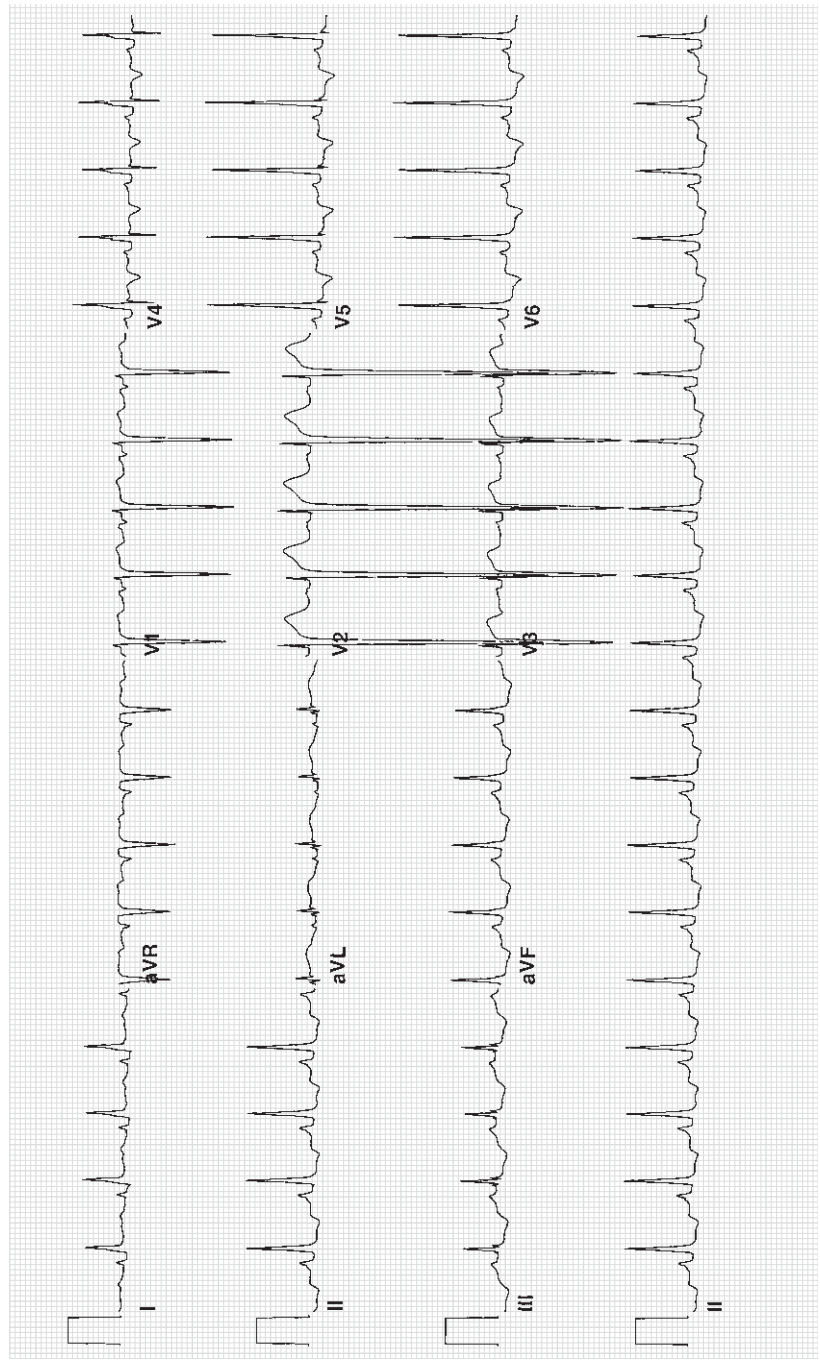


Figure 20 13-year-old boy with exertional chest pain.

Figure 20

- Sinus rhythm, 116 bpm
- S wave in V2 = 45 mm
- R wave in V6 = 22 mm
- T wave inversion in leads II, III, aVF, V4–V6

Left ventricular hypertrophy with strain

- Criteria for left ventricular hypertrophy plus QRS–T angle $>90^\circ$
- ST depression with upward convexity and T-wave inversion in left precordial leads
- Reciprocal changes in right precordial leads: ST segment elevation, tall T waves

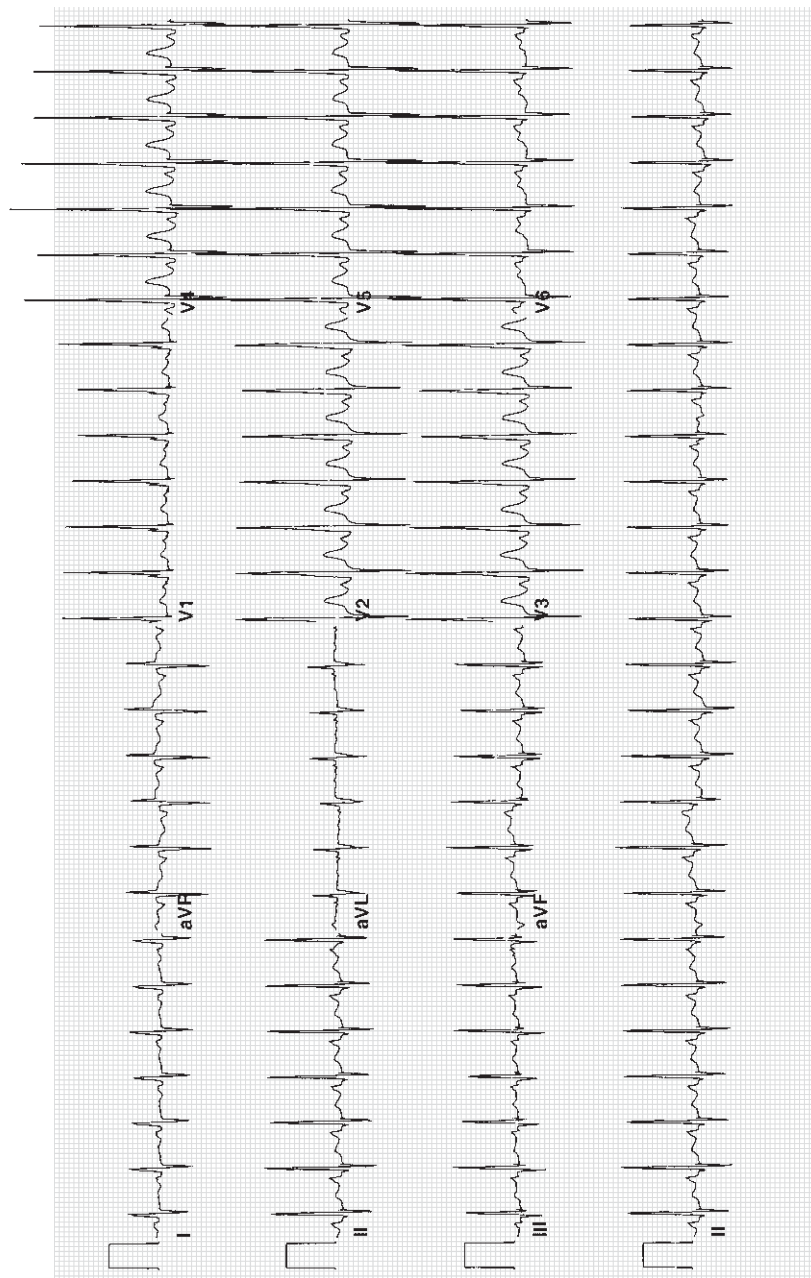


Figure 21 Neonate with critical aortic stenosis.

Figure 21

- Sinus tachycardia, 160 bpm
- rR' pattern in V1, R' = 20 mm
- Upright T wave in lead V1
- S wave in V6 = 9 mm
- R wave in V6 = 27 mm
- T wave strain pattern in V6

Biventricular hypertrophy

- Criteria for both right ventricular hypertrophy and left ventricular hypertrophy are met
- Right ventricular hypertrophy:
 - R wave in V1 >98th percentile
 - S wave in V6 >98th percentile
- Left ventricular hypertrophy:
 - R wave in V6 >98th percentile

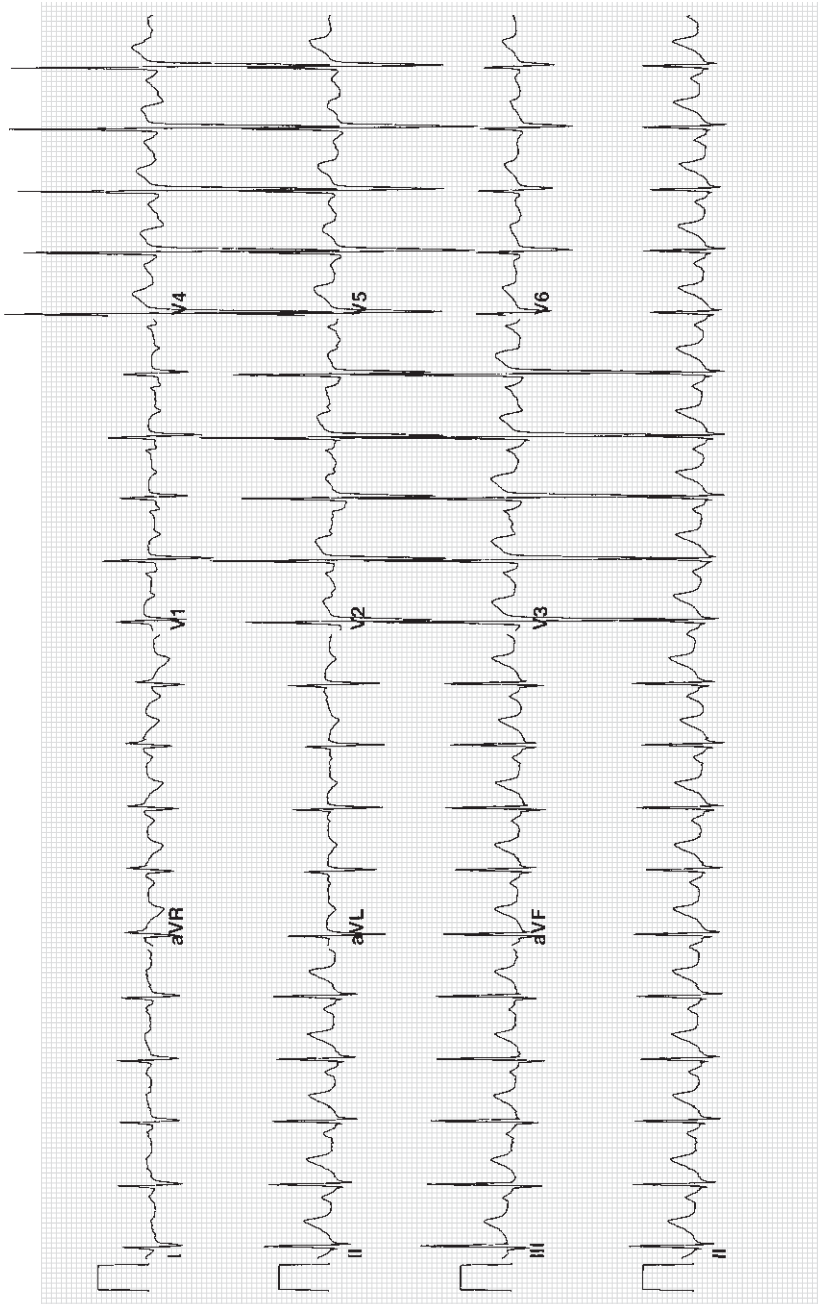


Figure 22 1-year-old female with ventricular septal defect.

Figure 22

- Normal sinus rhythm, 120 bpm
- Broad P wave in lead II
- R + S wave amplitudes in V3 >65 mm
- S wave in V6 $\geq$ 7 mm

Biventricular hypertrophy: 1 year old

- “Katz–Wachtel” criteria: R + S wave in lead V4 >60 mm
- Use cautiously without other supportive criteria for left ventricular hypertrophy and right ventricular hypertrophy

Reference

Katz LN, Wachtel H. The diphasic QRS type of electrocardiogram in congenital heart disease. *Am Heart J* 1937;13:202–206.

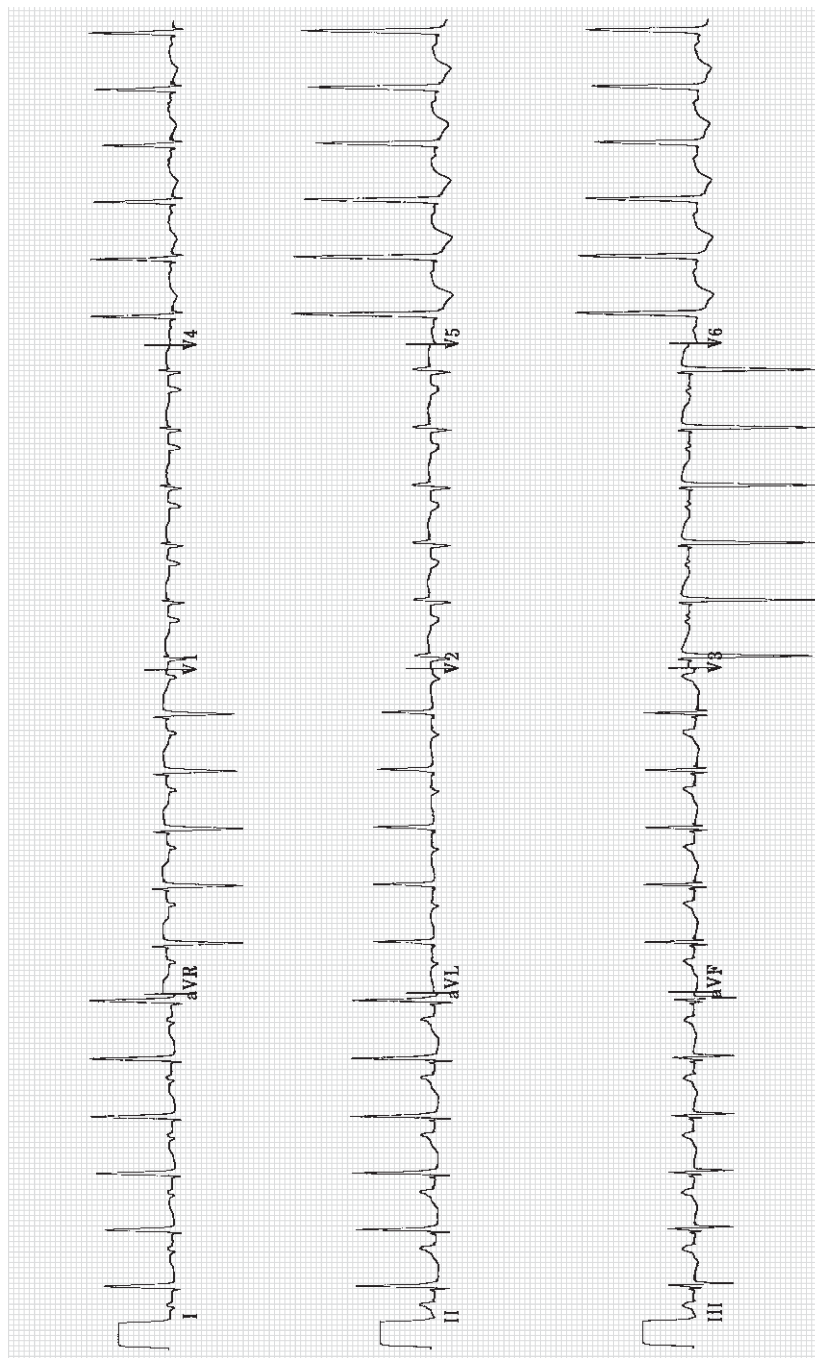


Figure 23 15-year-old male with progressive fatigue.

Figure 23

- Sinus tachycardia, 135 bpm
- Deep negative P wave in V1: left atrial enlargement
- Q wave in V1, V2
- Poor R wave progression in V1–V3
- T wave inversion in V4–V6 with wide QRS–T angle: left ventricular strain pattern

Dilated cardiomyopathy

- ECG findings are nonspecific
- Left atrial enlargement in >30%
- Left ventricular hypertrophy in >50%, often with ventricular strain pattern
- Low voltage QRS in limb leads
- Intraventricular conduction delay, or left bundle branch block 10–20%
- Abnormal ST, T waves >50%
- Supraventricular and ventricular arrhythmias common

References

- Friedman RA, Moak JP, Garson A, Jr. Clinical course of idiopathic dilated cardiomyopathy in children. *J Am Coll Cardiol* 1991;8:152–156.
- Arola A, Tuominen J, Ruusken O, Jokinen E. Idiopathic dilated cardiomyopathy in children: prognostic indicators and outcome. *Pediatrics* 1998;101:369–376.

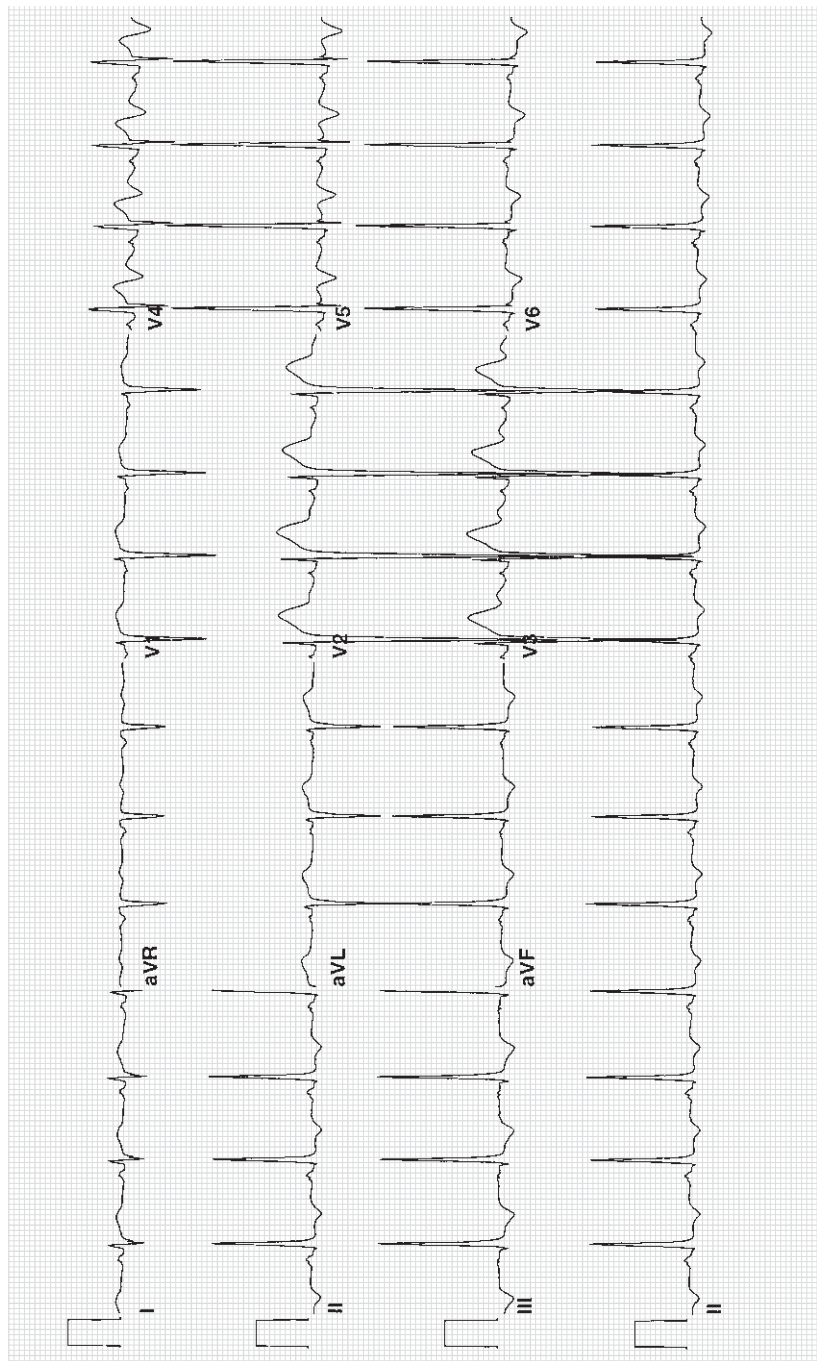


Figure 24 13-year-old male athlete, status post-cardiac arrest during sports.

Figure 24

- Normal sinus rhythm, 93 bpm
 - Broad, notched P wave suggests left atrial enlargement
 - Left ventricular hypertrophy with strain:
 - R wave in V6 = 27 mm
 - S wave in V2 >40 mm
 - T wave inversion in II, III, aVF, V4–V6
- QRS–T wave angle discordance

Hypertrophic cardiomyopathy

- Left axis deviation in 10–30%
- Left atrial enlargement common
- Left ventricular hypertrophy 50–65%
- Abnormal Q waves in anterolateral leads (I, aVL, V4–V6) in 20–30%
- ST and T wave abnormalities very common

Reference

Charron P, Dubourg O, Desnos M, Bouhour JB, Isnard R, Hagege A, Carrier L, Bonne G, Tesson F, Richard P, Hainque B, Schwartz K, Komajda M. Diagnostic value of electrocardiography and echocardiography for familial hypertrophic cardiomyopathy in genotyped children. *Eur Heart J* 1998;19(9):1377–1382.

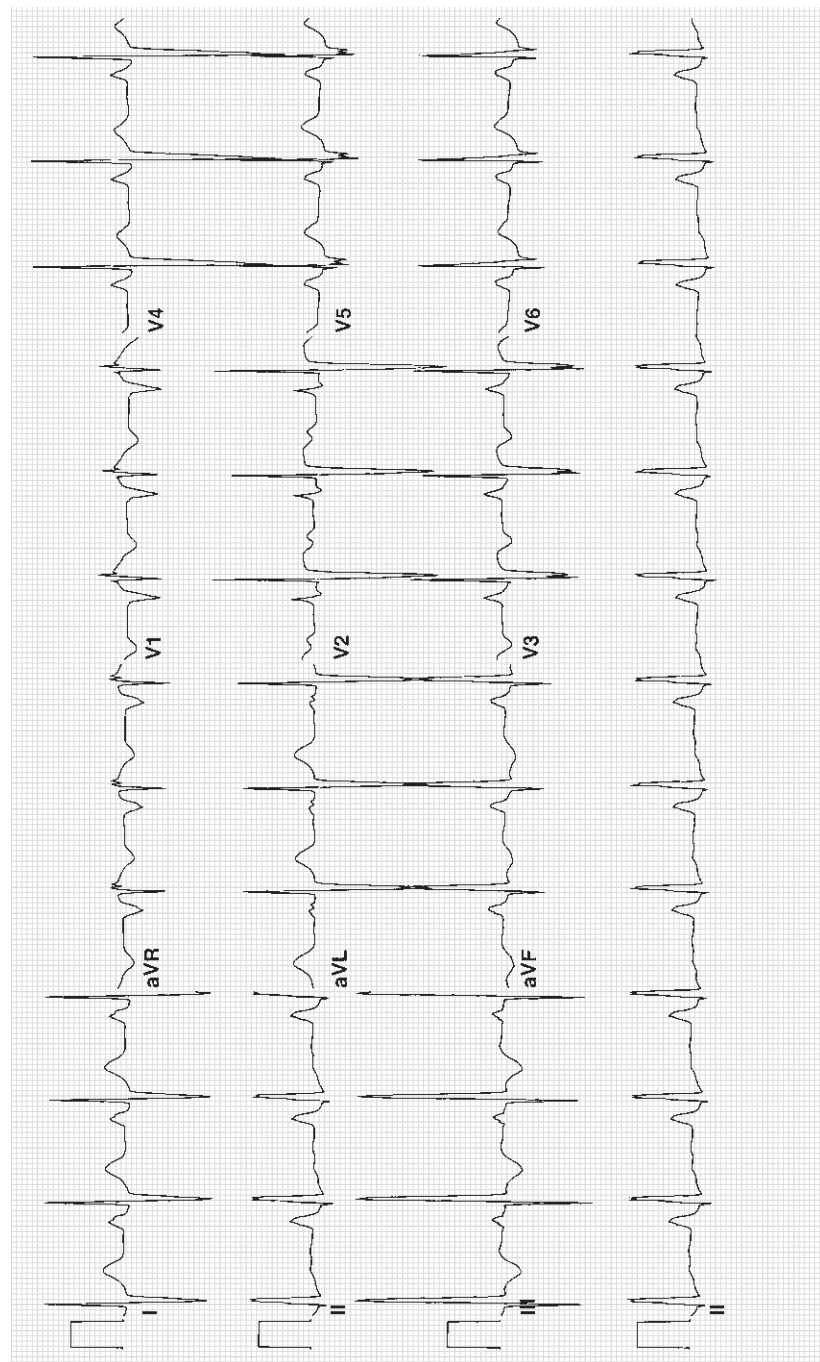


Figure 25 7-year-old girl evaluated for recurrent syncope.

Figure 25

- Sinus rhythm, 75 bpm
- Batrial enlargement:
Tall, peaked and broad P waves in II, aVF, V2–V3
Deep, broad negative P wave in V1
- QRS duration = 0.10 sec: mild left ventricular conduction delay
- Left ventricular hypertrophy:
R wave in III = 28 mm
S wave in V2 = 21 mm
Q wave in III = 12 mm

Restrictive cardiomyopathy

- Marked biatrial enlargement, often in absence of ventricular hypertrophy
- QRS and ST/T wave abnormalities may be seen

Reference

Rivenes SM, Kearney DL, Smith O'B, Towbin JA, Denfield SW. Sudden death and cardiovascular collapse in children with restrictive cardiomyopathy. *Circulation* 2000;102:876–882.

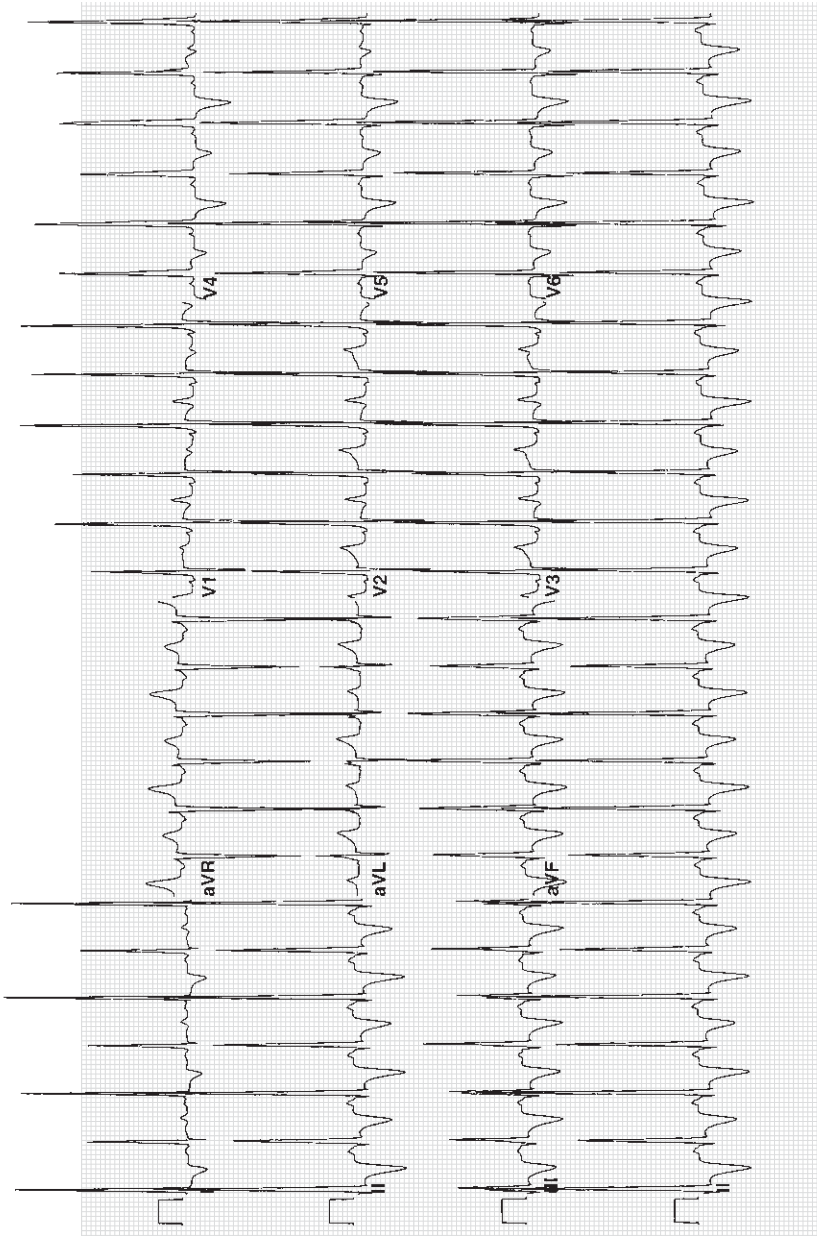


Figure 26 1-year-old boy with hypotonia.

Figure 26

- Sinus rhythm, 146 bpm
- Short PR interval = 0.08 sec
- QRS and P alternans, best seen in aVL and V1
- Biventricular hypertrophy with strain:
 - R wave in V1 ≥ 25 mm
 - R wave in V6 > 29 mm
 - Q wave in V6 = 9 mm
 - Deep T wave inversion in II, III, aVF, V4–V6

Pompe's Disease

- Short PR interval
- Marked biventricular hypertrophy
- QRS, ST changes
- Alternans:
 - Alternation in configuration or magnitude of P, QRS complexes, and/or T waves
 - Associated with pericardial effusion, supraventricular tachycardia

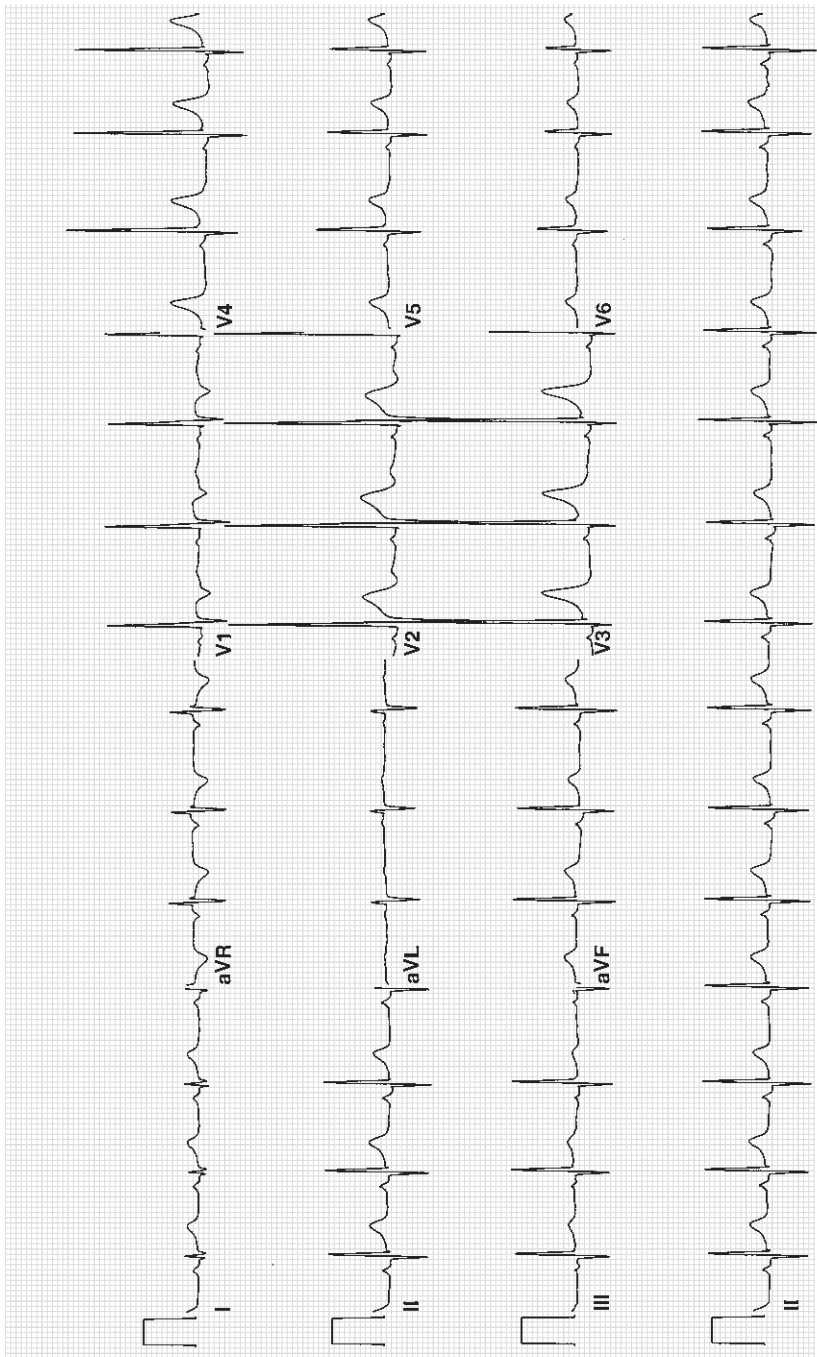


Figure 27 8-year-old boy with muscular dystrophy.

Figure 27

- Sinus rhythm, 84 bpm
- Right ventricular hypertrophy
R wave in V1 = 18 mm
- Deep Q waves in leads II, III, aVF, V4–V6 suggest left ventricular hypertrophy

Duchenne's muscular dystrophy

- Sinus tachycardia very common
- PR interval short or normal
- Ventricular hypertrophy common
- Deep narrow Q waves may mimic infarct pattern, often seen in leads I, aVL, V5–V6
- Associated conduction defects common and progressive

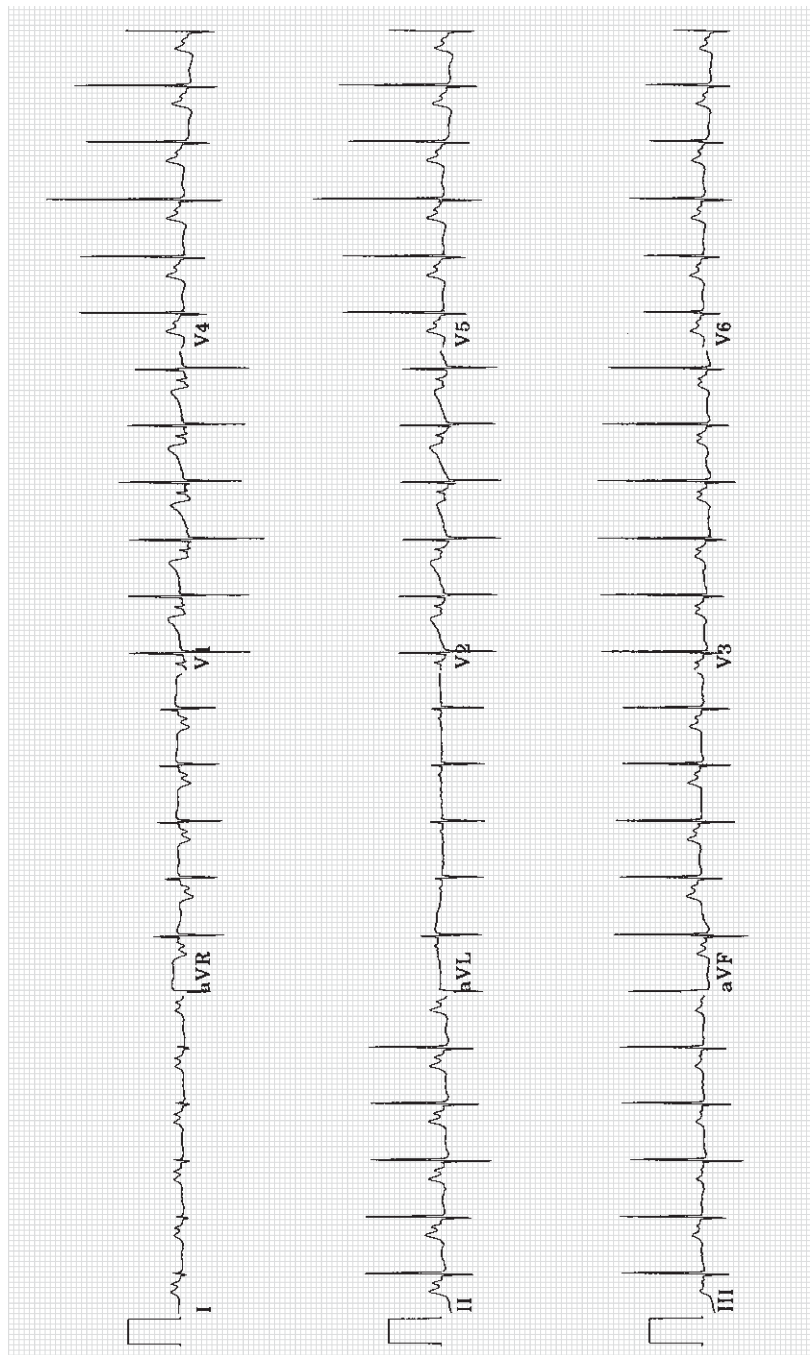


Figure 28 2-month-old baby with marked hypocalcemia. (ECG courtesy of Richard Friedman, M.D.)

Figure 28

- Sinus rhythm, 136 bpm
- Right ventricular hypertrophy: upright T wave in V1
- Marked QT prolongation:
 $QTc = 0.55 \text{ sec}$
 T wave ends at onset of P wave
- Nonspecific ST wave changes

Hypocalcemia

- QT prolongation due to prolonged ST segment
- Low amplitude T waves; normal T wave duration
- Degree of prolongation from onset of QRS complex to onset of T wave correlates with severity of hypocalcemia

Reference

Colletti RB, Pan MW, Smith EWP, Genel M. Detection of hypocalcemia in susceptible neonates: The Q-oTc interval. *N Engl J Med* 1974;290:931–935.

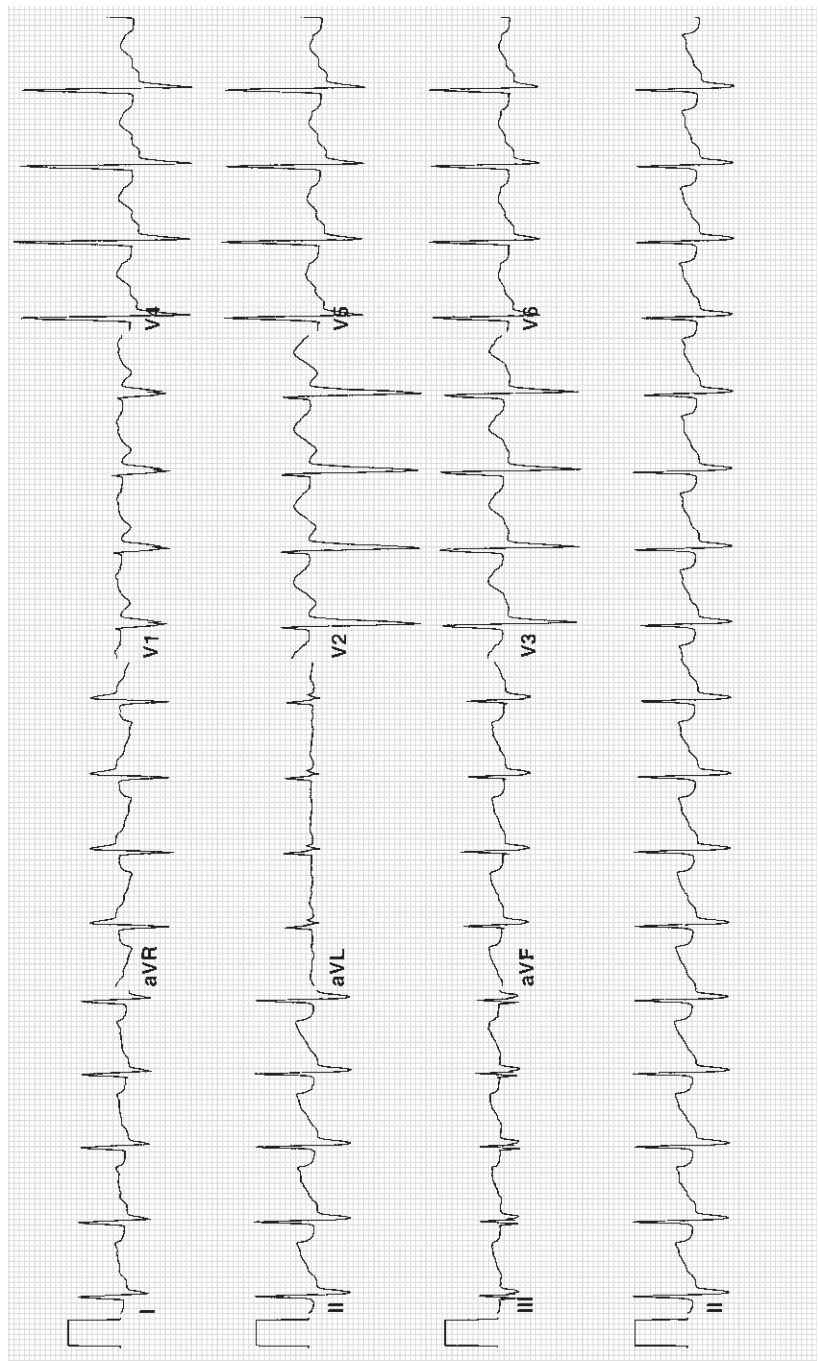


Figure 29 13-year-old girl with familial periodic paralysis.

Figure 29

- Sinus rhythm, 104 bpm
- First degree atrioventricular block: PR interval = 0.18 sec
- Intraventricular conduction delay: QRS duration = 0.11 sec
- Broad and flattened T waves
- Diffuse ST and T wave abnormalities

Hypokalemia

- Decreased T wave amplitude
- ST segment depression
- Prominent U waves >1 mm, may be taller than T wave
- Normal QTc (presence of U wave makes measurement difficult)
- QRS prolongation in advanced hypokalemia
- Arrhythmias (especially in presence of digoxin): atrial tachycardia with block; ventricular arrhythmia: 1°, 2°, and 3° atrioventricular block

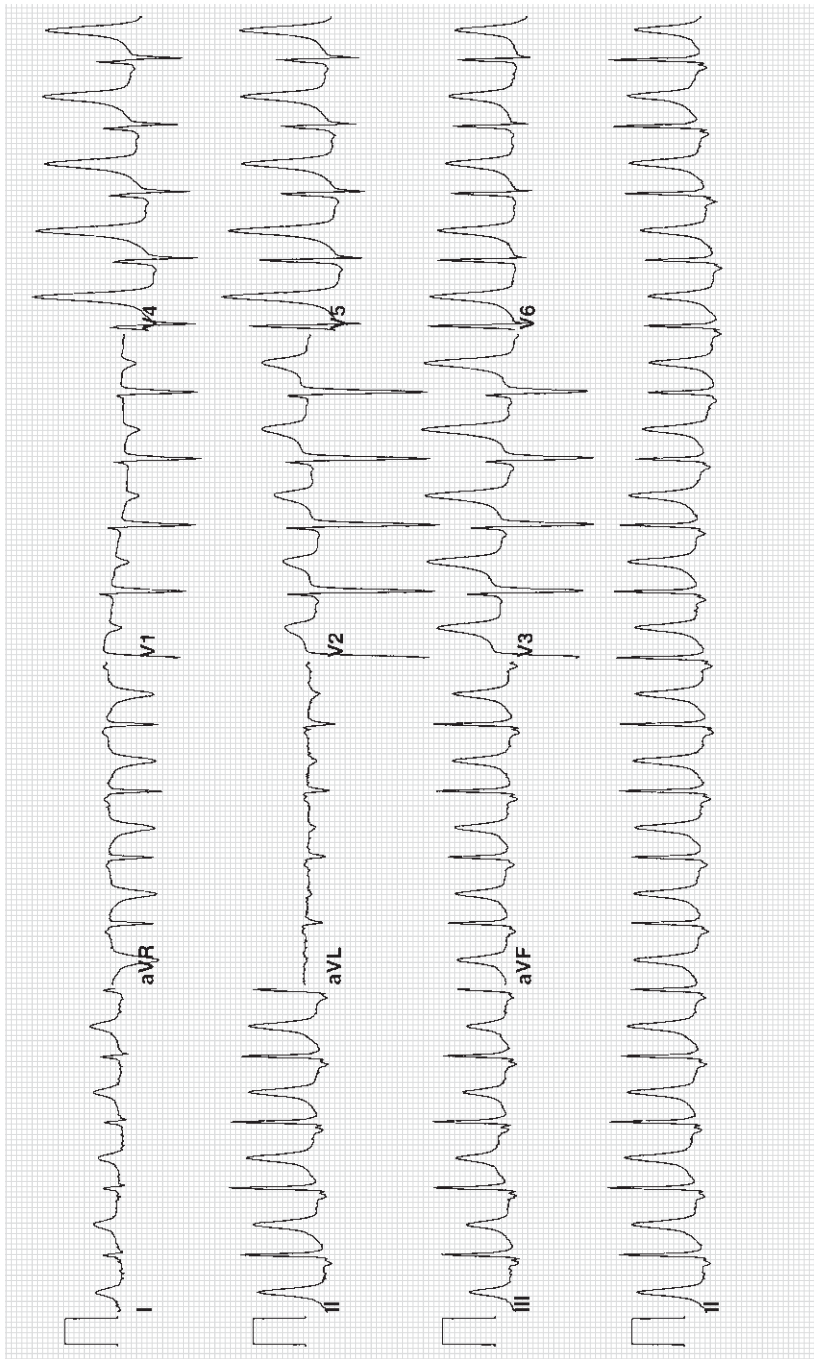


Figure 30 Young adult with renal failure, marked hyperkalemia ($K^+ = 7.4 \text{ mEq/L}$).

Figure 30

- Atrial tachycardia, 120bpm with low amplitude, inverted P waves in II, III, aVF: low atrial focus
- Giant peaked T waves
- QT prolongation: QTc = 0.54 sec

Hyperkalemia

- Progressive changes as K⁺ increases:
- Tall, narrow T waves; “pinch-bottomed” or tented: K⁺ >5.5 mEq/L
 - PR prolongation, ST segment depression, QRS prolongation (intraventricular conduction delay): K⁺ >6.5 mEq/L
 - P wave amplitude decreases (K⁺ >7.0 mEq/L) or absence of P wave (K⁺ >8.5 mEq/L)
 - ST segment elevation simulates ischemia
 - Arrhythmias: Atrioventricular block, junctional or ventricular rhythm, ventricular tachycardia or ventricular fibrillation

Reference

Mattu A, Brady WJ, Robinson DA. Electrocardiographic manifestations of hyperkalemia. *Am J Emerg Med* 2000;18(6):721–729.

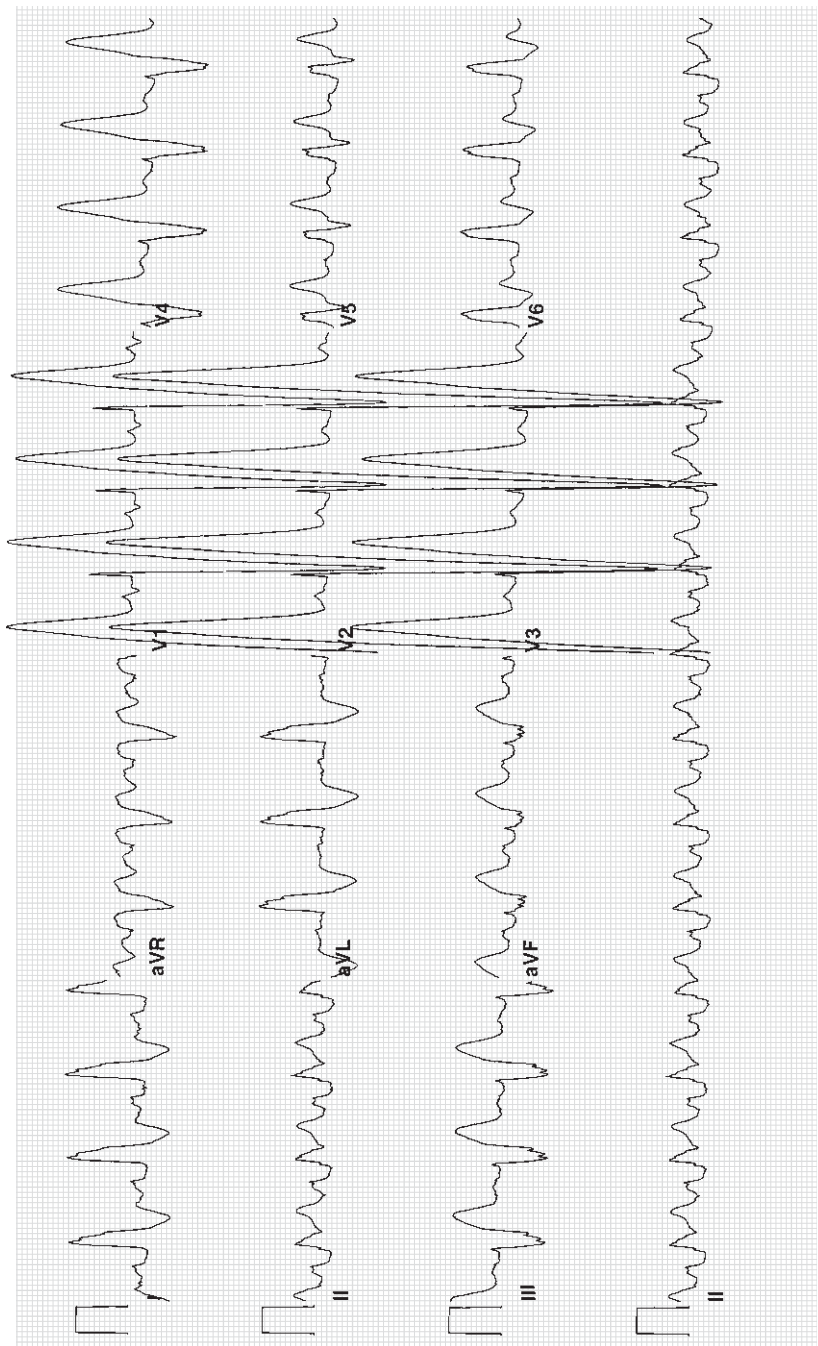


Figure 31 17-year-old male with renal failure.

Figure 31

- Sinus rhythm, 92 bpm
- First degree atrioventricular block:
PR interval = 0.21 sec
- Broad notched P waves
- Left bundle branch block:
QRS duration = 0.16 sec
- Marked QT prolongation, QTc = 0.52 sec
- Tall, broadened T waves
- Diffuse ST abnormalities

Hyperkalemia

Advanced changes:

- First degree atrioventricular block
- QRS prolongation
- Arrhythmias:
Absence of P wave; atrioventricular block; ventricular tachycardia or fibrillation

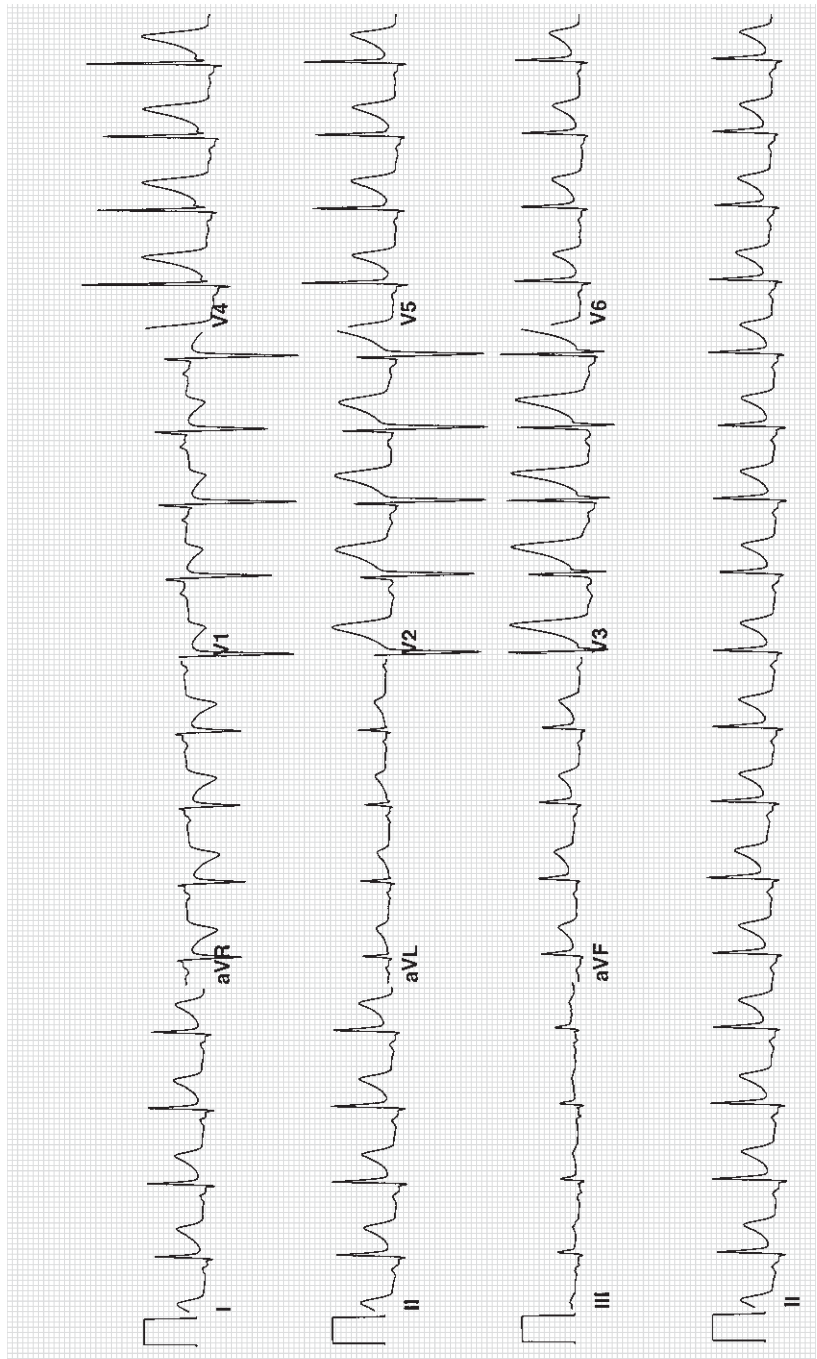


Figure 32 10-year-old boy with autoimmune collagen vascular disease, presents with chest pain.

Figure 32

- Sinus rhythm, 105 bpm
- PR segment depression in leads I, II, aVF, V4–V6
- Diffuse ST segment elevation, best seen in leads I, II, aVF, V2–V6

Pericarditis

Progressive ECG changes over time:

- Diffuse ST segment elevation
- T wave changes: flat, followed by biphasic, or inverted
- PR segment depression
- With large pericardial effusion:
 - Low voltage QRS
 - Electrical alternans of QRS

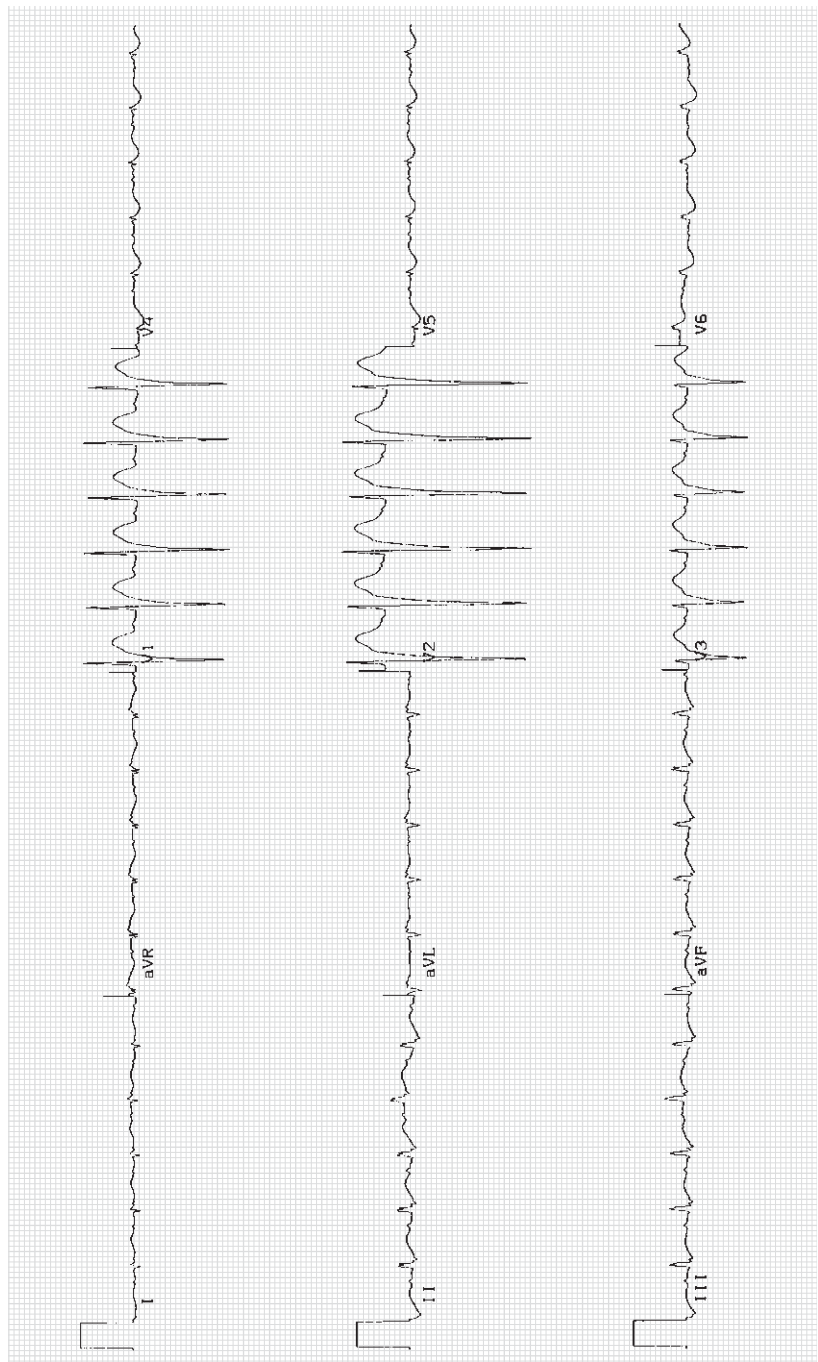


Figure 33 13-day-old infant with intrauterine infection and cardiomegaly.

Figure 33

- Sinus rhythm, 140 bpm
- Low amplitude P waves and QRS complexes in limb leads
- Poor R wave progression in precordial leads
- Low amplitude QRS in V4–V6
- Deep S wave in V2
- Diffuse ST segment and T wave abnormalities

Myocarditis

Highly variable findings:

- Sinus tachycardia
- Generalized low voltage QRS complexes: <5 mm in limb leads, or <8 mm in precordial leads
- Q waves, poor R wave progression across left precordium
- ST segment changes
- T wave flattening or inversion
- Arrhythmias: atrial tachycardia, atrioventricular block, and ventricular arrhythmias

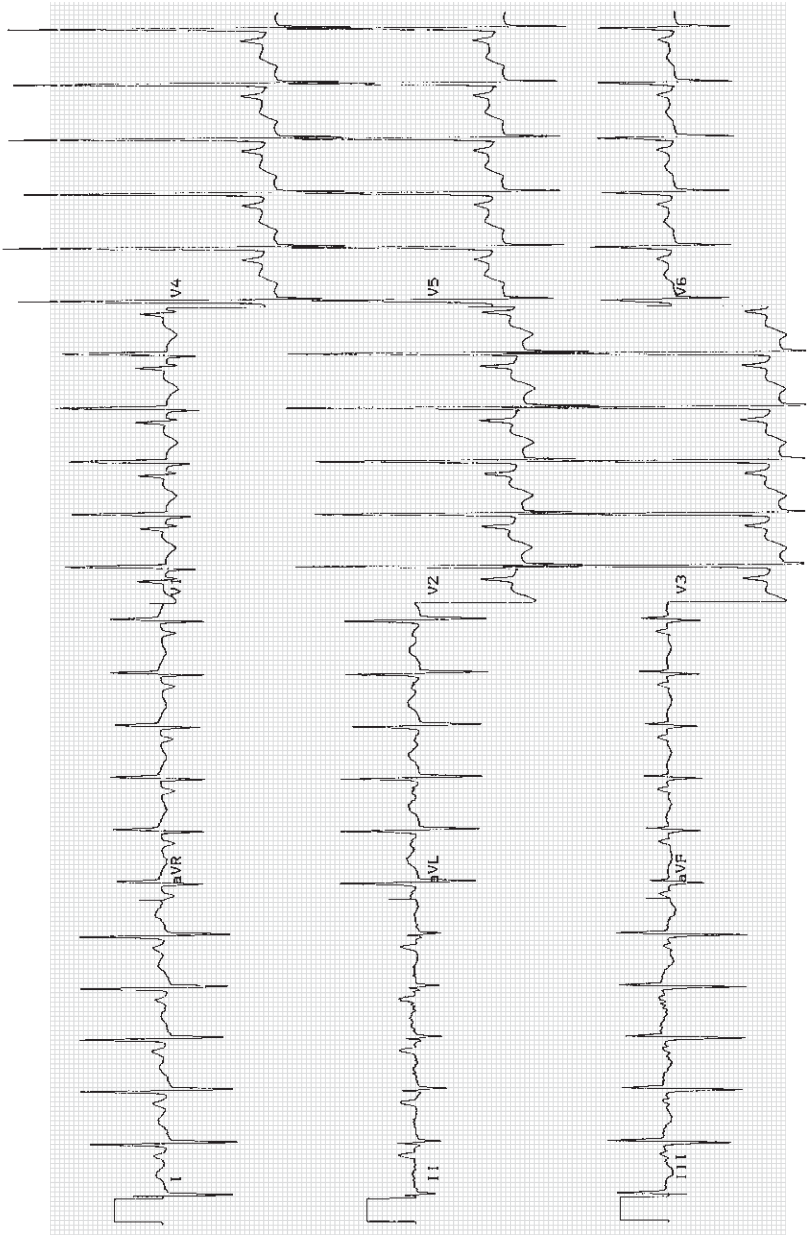


Figure 34 2-year-old boy with severe pulmonary hypertension.

Figure 34

- Sinus rhythm, 135 bpm
- Left axis deviation:
QRS axis = -60°
- Right atrial enlargement: tall, peaked P wave in leads II, V1–V4
- Q wave in leads III, aVF, and V1: septal hypertrophy
- Severe right ventricular hypertrophy with strain:
Q wave in V1
R wave in V1 = 20 mm
S wave in V6 = 12 mm
ST depression in V2–V5

Pulmonary hypertension

- Mild pulmonary hypertension:
ECG may be normal
- Increasing pulmonary hypertension:
Right axis deviation
Right atrial enlargement
Right ventricular hypertrophy
Right ventricular hypertrophy with strain

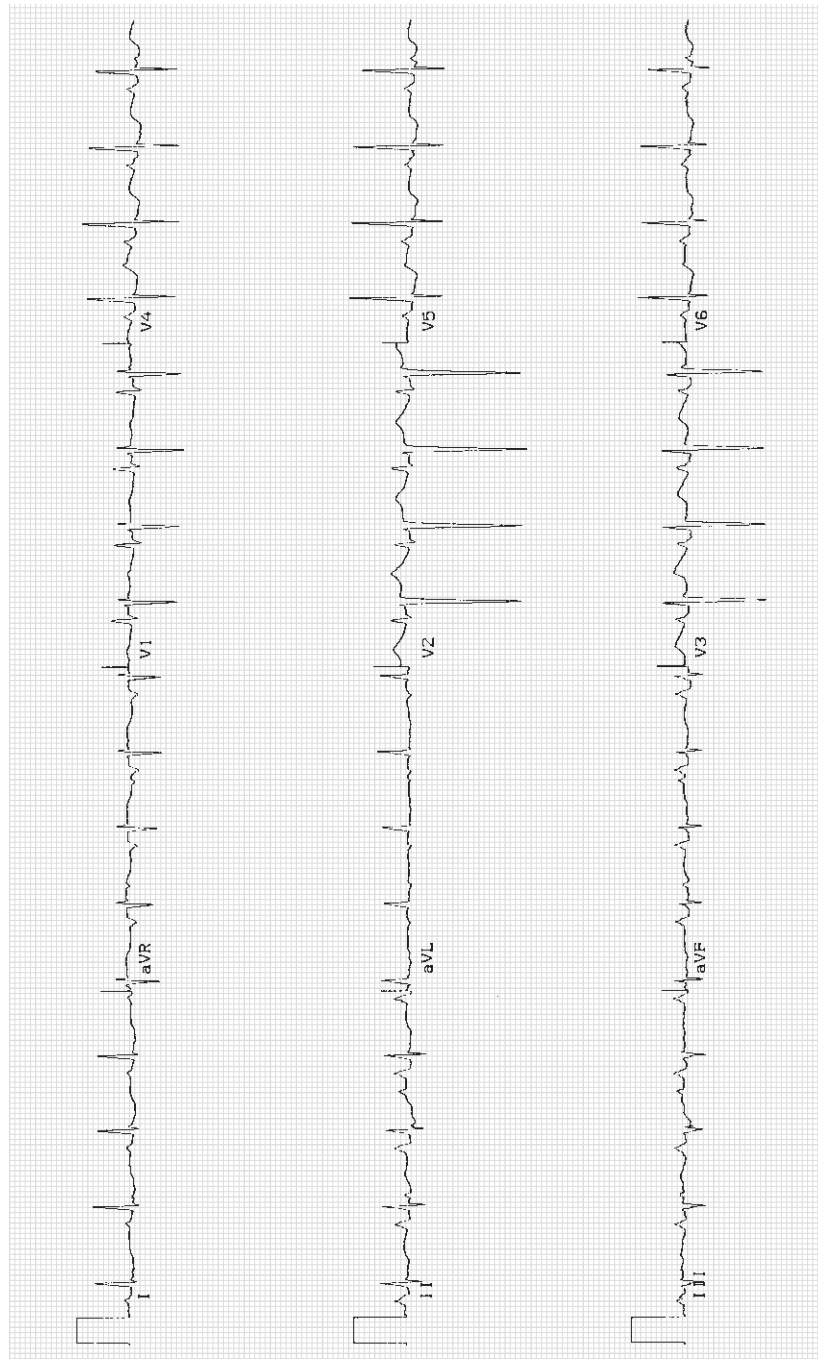


Figure 35 17-year-old male status post heart transplantation.

Figure 35

- Sinus rhythm, 100 bpm
- Two P waves of different morphologies, best seen in leads II, III, aVF
- Left axis deviation: QRS axis = -10°
- Right atrial enlargement: tall peaked P wave in V1–V2
- Diffuse nonspecific ST and T wave abnormalities

Heart transplantation

- Two sets of P waves: one from recipient, one from donor heart (not seen with bicaval anastomosis technique)
- Incomplete, or complete right bundle branch block common
- Left anterior hemiblock less common (results in left axis deviation)
- Arrhythmias: sinus node dysfunction, junctional rhythm, premature atrial or ventricular complexes: may reflect acute rejection or coronary artery disease

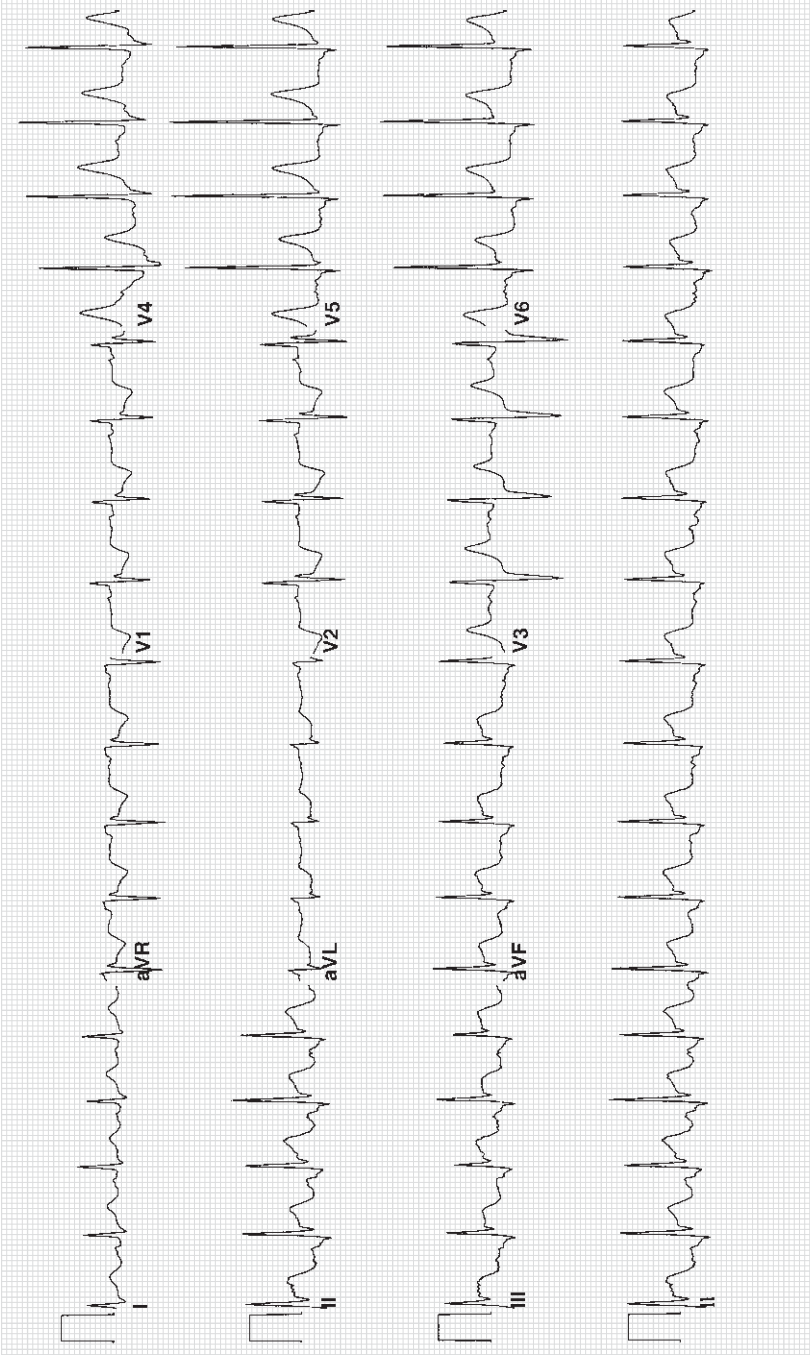


Figure 36 8-year-old girl with onset of acute chest pain the morning after radiofrequency catheter ablation of right posteroseptal accessory connection.

Figure 36

- Sinus rhythm, 105 bpm
 - Notched P wave suggests left atrial enlargement
 - Marked ST elevation in leads II, III, aVF, V6: inferolateral ischemia
 - ST depression in leads aVL, V1–V4
 - Left ventricular hypertrophy:
 - R wave in V6 = 28 mm
-
- Acute myocardial ischemia**
- ST segment elevation (“current of injury”) reflects distribution of affected myocardium:
 - Inferior wall: II, III, aVF
 - Lateral wall: I, aVL, V6
 - Anterior wall: V1–V5
 - Reciprocal ST segment depression
 - T wave changes:
 - Hyperacute phase: tall, peaked
 - Subsequent: T wave inversion
 - Later appearance of pathological Q waves indicates transmural myocardial infarction

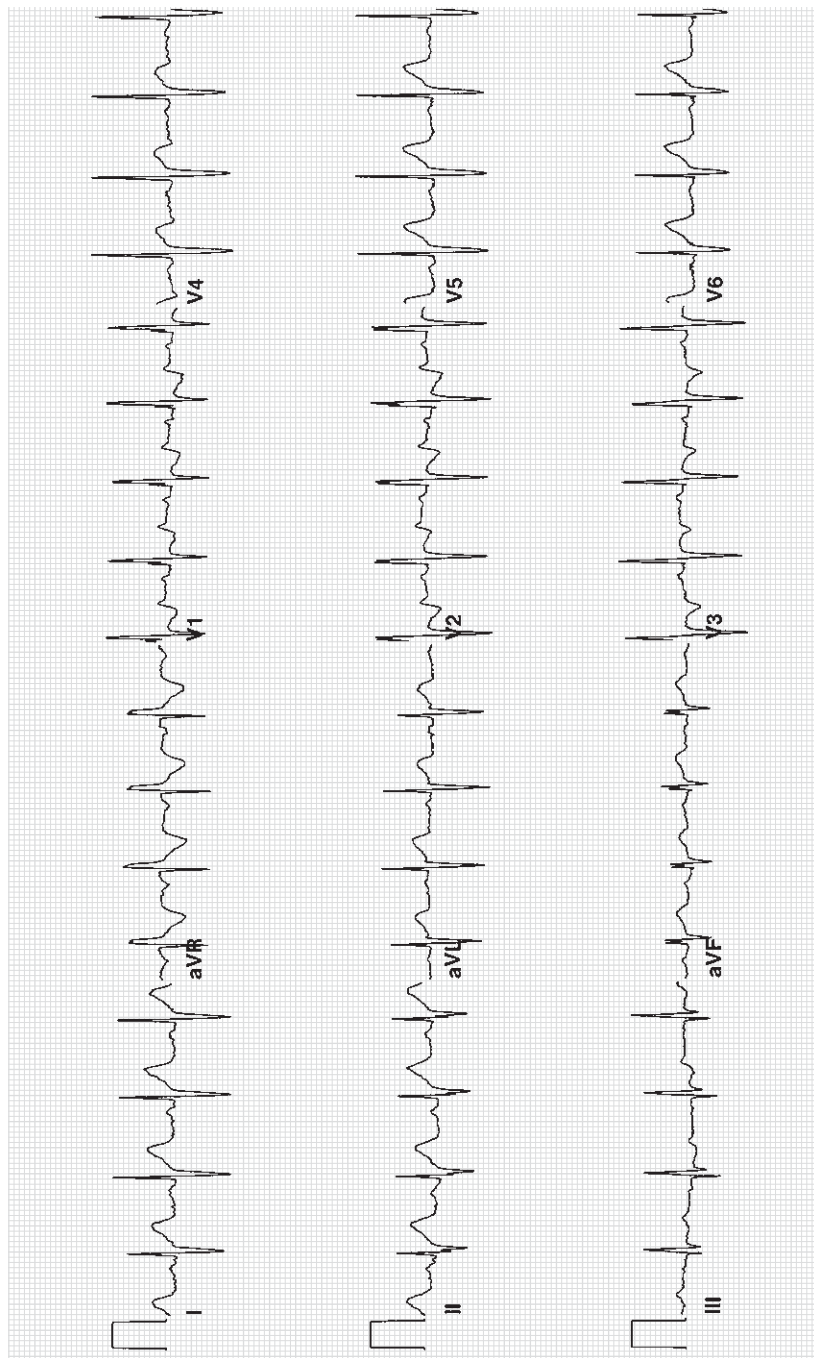


Figure 37 3-year-old girl referred with systolic murmur at left upper sternal border and fixed splitting of second heart sound.

Figure 37

- Sinus rhythm, 104 bpm
- Right ventricular conduction delay pattern: rsR' pattern in V1; R' is greater than twice the height of r wave
- R/S ratio >1 in V1, and S wave in V6 = 8 mm, consistent with mild right ventricular hypertrophy

Atrial septal defect, secundum type

- QRS axis often rightward, usually $>+90^\circ$
- rsR', rSr' pattern in V1 seen in 60–90%
- Right atrial enlargement in up to 50%
- Prolonged PR interval in 10–20%
- ECG may be normal in up to 5% of patients
- Increasing incidence of supraventricular tachycardia (atrial flutter/fibrillation) seen in $>30\%$ of patients after age 40 years

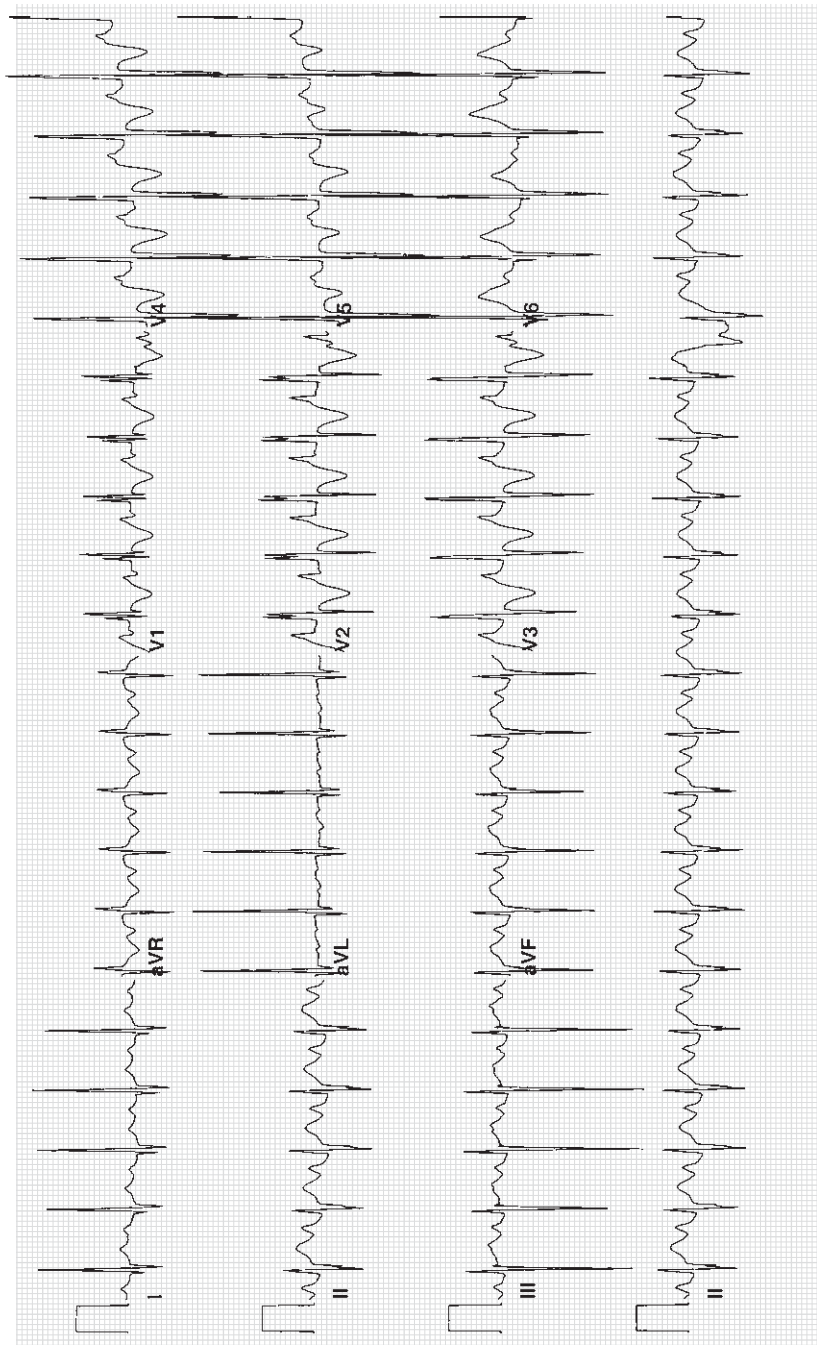


Figure 38 3-year-old boy with murmur and cardiomegaly.

Figure 38

- Sinus rhythm, 130 bpm
- Left axis deviation: QRS axis -40°
- First degree atrioventricular block:
PR interval = 0.180 sec
- Right atrial enlargement; tall peaked P waves
- rsR' in V1
- Left ventricular hypertrophy: R wave in V6 = 34 mm
- Right ventricular hypertrophy: S wave in V6 >15 mm

Atrial septal defect, primum type

- Leftward QRS axis, superior to -30° , in 90%
- First degree atrioventricular block in 20–50%
- Right and left atrial enlargement common
- rsR' pattern typically seen in V1
- Right and left ventricular hypertrophy

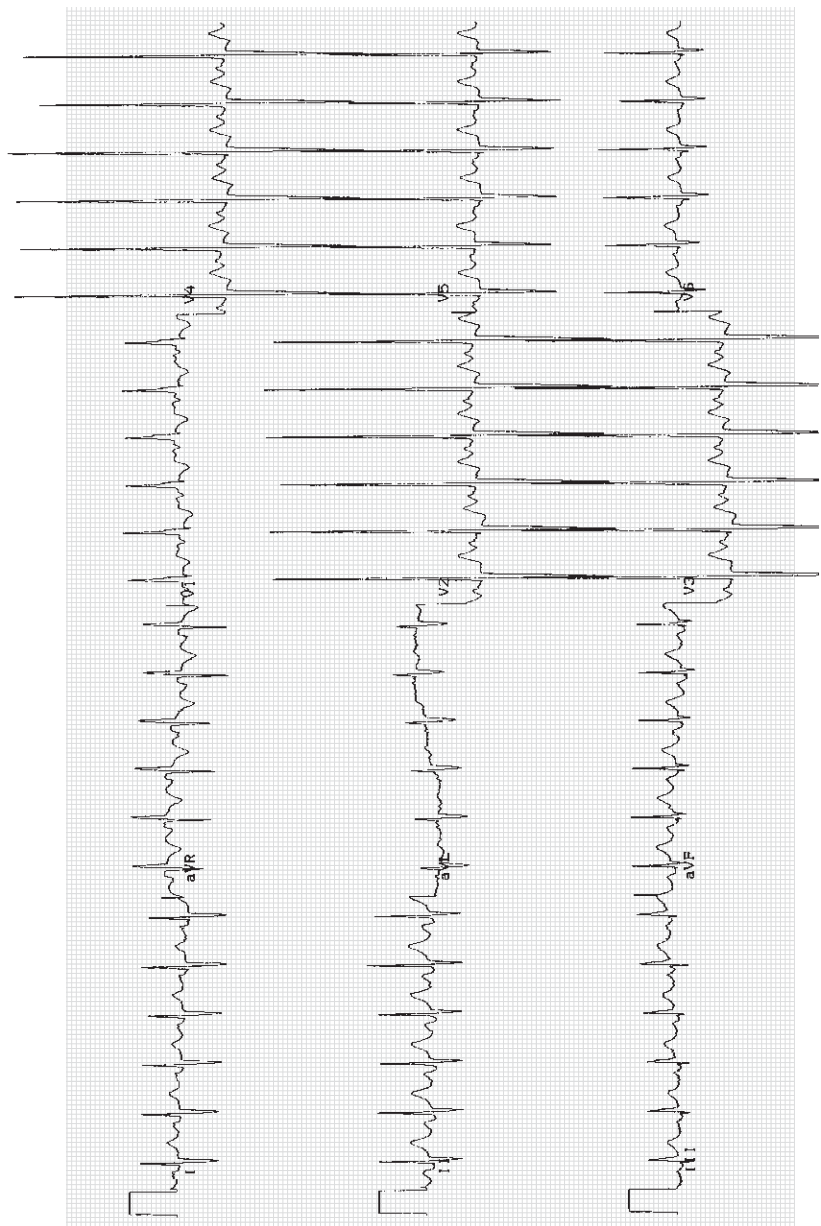


Figure 39 2-month-old infant with murmur; poor weight gain.

Figure 39

- Sinus rhythm, 145 bpm
- Possible left atrial enlargement
 - Notched P wave in I, V5
 - Prominent negative component of P wave in V1
- Biventricular hypertrophy
 - R + S wave amplitudes in V2 = 70 mm
 - R + S wave amplitudes in V4 > 65 mm

Ventricular septal defect: infant 2 months old

- QRS axis usually $+90^\circ$ to $+150^\circ$
- Occasional left axis deviation with perimembranous defect
- First degree atrioventricular block in $<10\%$
- Left ventricular hypertrophy with significant left-to-right shunt
- Occasional right ventricular hypertrophy
- Infants: biventricular hypertrophy with midprecordial voltage >60 mm
- Large Q waves in leads II, III, aVF, V6 reflect septal hypertrophy

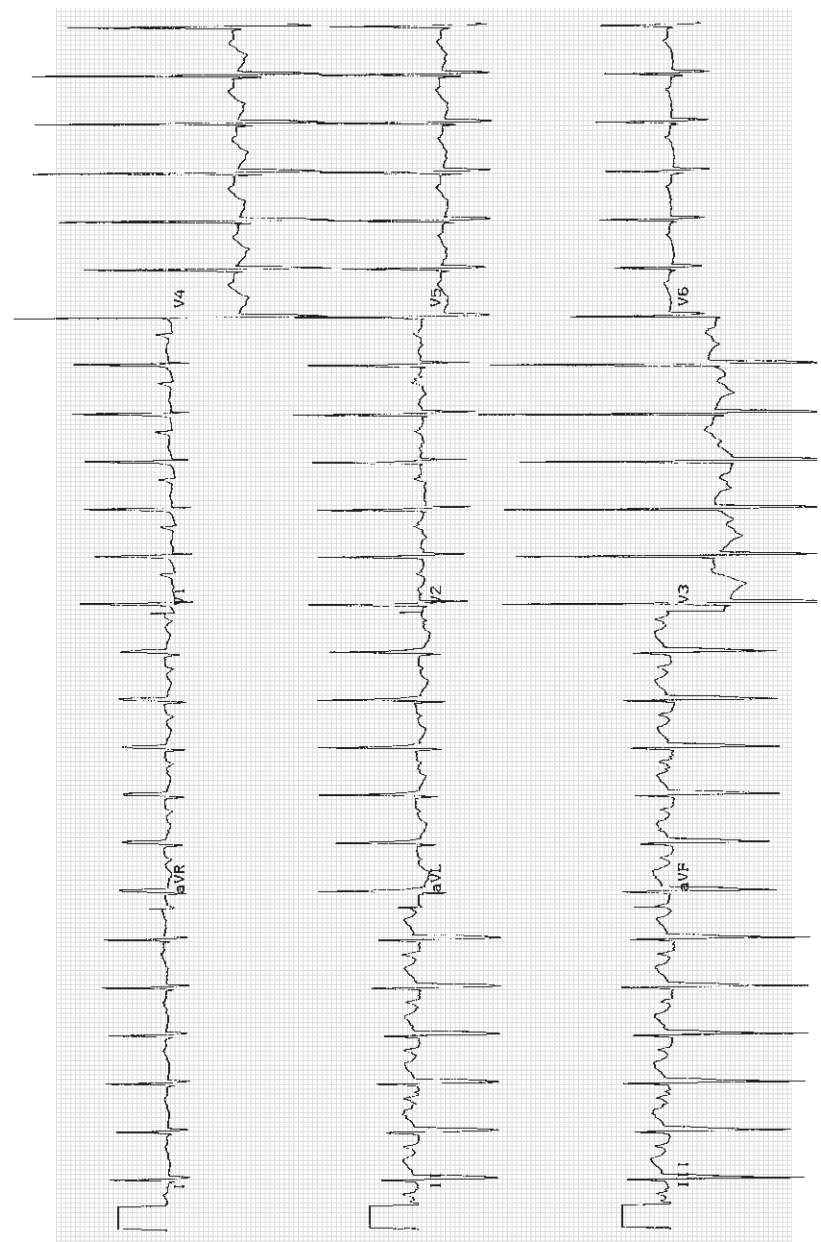


Figure 40 2-month-old infant with Down syndrome, murmur, and cardiomegaly; failure to thrive.

Figure 40

- Sinus rhythm, 145 bpm
- Left axis deviation: QRS axis -60°
- First degree atrioventricular block: PR interval = 0.15 sec
- Right atrial enlargement: peaked P waves
- Right ventricular hypertrophy:
 - R wave in V1 = 18 mm
 - Upright T wave in V1
 - S wave in V6 = 7 mm
- Left ventricular hypertrophy
 - R wave in V5 = 25 mm
- Left ventricular strain: T wave inversion in V4–V6

Atrioventricular septal defect, complete

- Superior or left axis deviation $>-30^\circ$ in 95%
- Prolonged PR interval in 50%
- Atrial enlargement in $>50\%$
- Right ventricular hypertrophy common, with rSR' or RSR' in lead V1
- Left ventricular hypertrophy with large shunts

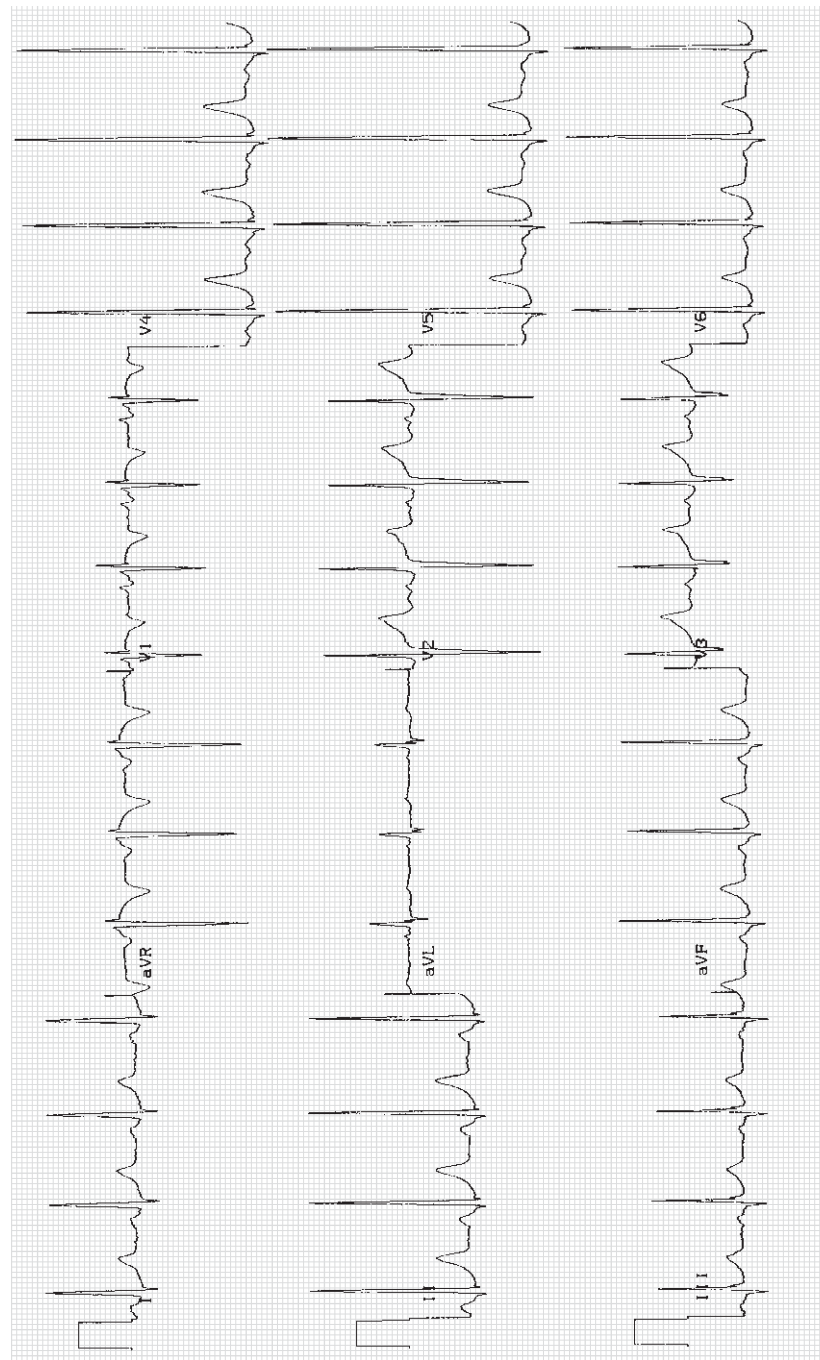


Figure 41 8-year-old girl with continuous murmur in infravalvular area; mild cardiomegaly.

Figure 41

- Sinus rhythm, 90 bpm
- Left atrial enlargement:
 - Broad P wave in I, II, V5, V6
 - Deep and wide negative terminal component of P wave in V1
- Left ventricular hypertrophy: R wave in V6 = 34 mm
- Early repolarization

Patent ductus arteriosus (PDA)

- Normal ECG with small PDA
- Normal QRS axis
- Left ventricular hypertrophy: deep Q, tall R wave in leads II, III, aVF, V5–V6
- Left atrial enlargement occasional
- ± Right ventricular hypertrophy

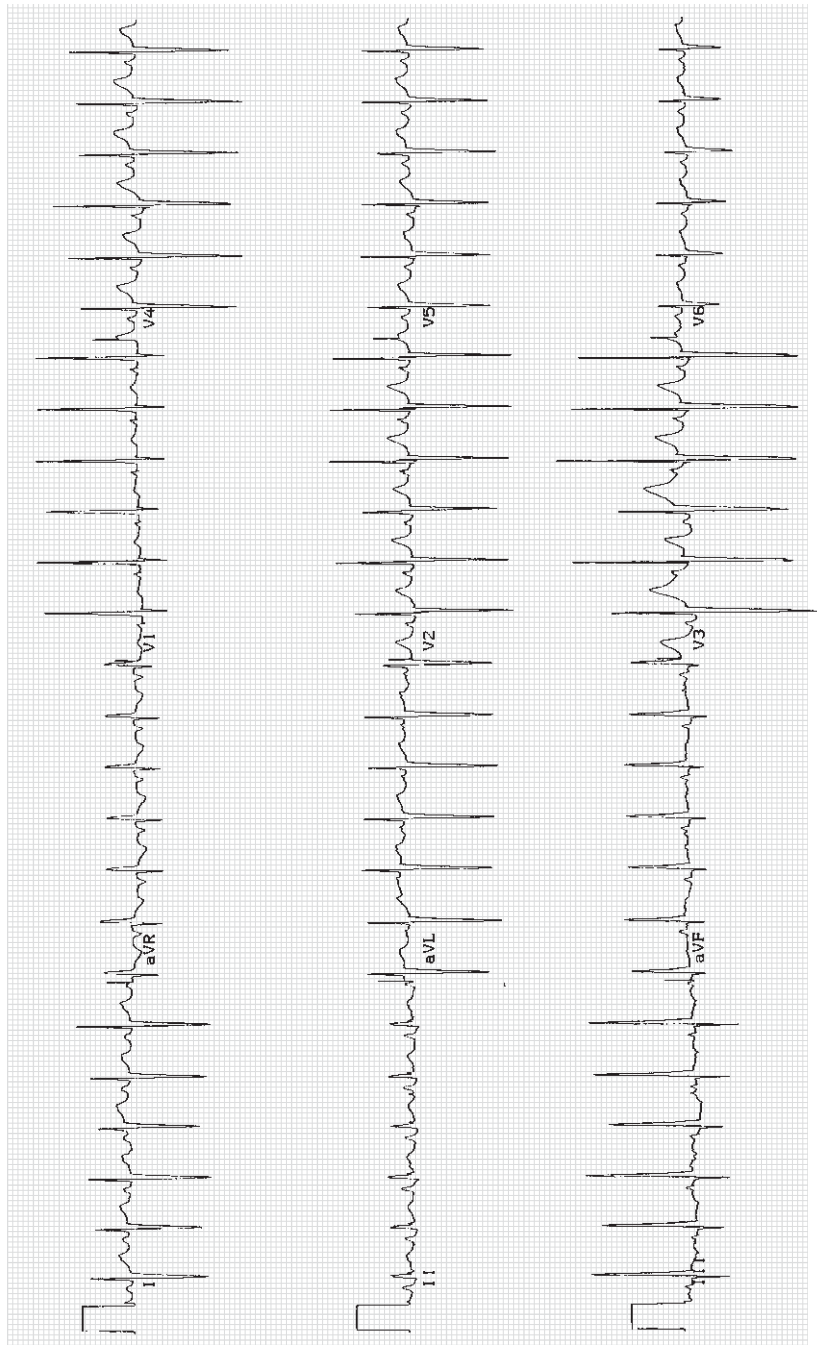


Figure 42 4-month-old infant with cyanotic spells and loud murmur at left sternal border.

Figure 42

- Sinus rhythm, 150 bpm
- Right axis deviation: QRS axis $+130^\circ$
- Right ventricular hypertrophy:
 - R wave in V1 > 18 mm
 - Upright T wave in V1
 - S wave in V6 = 8 mm

Tetralogy of Fallot

- Right axis deviation: QRS axis $+90^\circ$ to $+180^\circ$
- Right atrial enlargement: 30–50% of older children
- Moderate to severe right ventricular hypertrophy, increasing with age
- Left ventricular hypertrophy may be seen, particularly in “pink” tetralogy with minimal pulmonary stenosis and left-to-right shunt

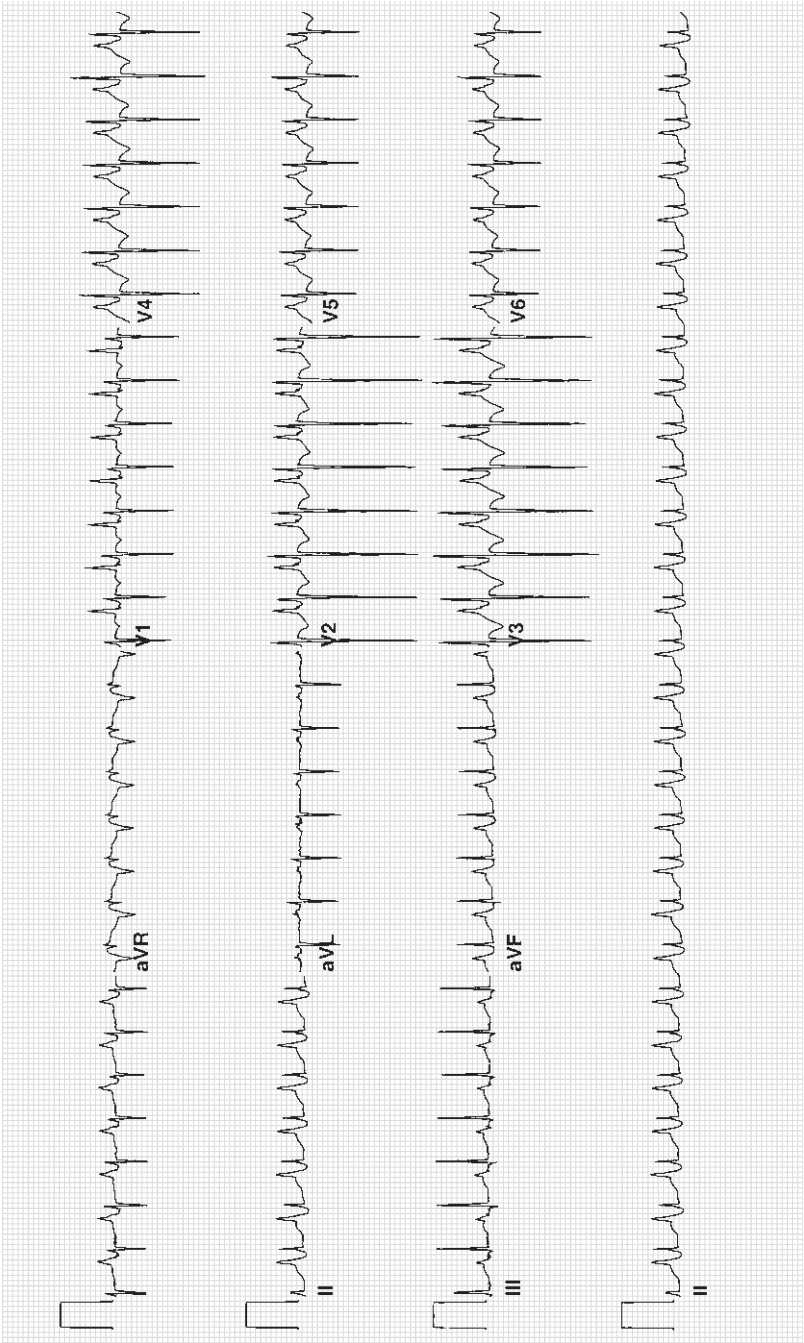


Figure 43 Neonate with severe cyanosis.

Figure 43

- Sinus rhythm, 180 bpm
- Right atrial enlargement, marked
- Decreased R wave amplitude in leads V1–V2
- Absent Q wave in V6: similarity of leads V4–V6 suggests abnormal lead placement

Pulmonary atresia with intact ventricular septum

- QRS axis: normal or leftward
- Right atrial enlargement
- Decreased right ventricular voltage
- Left ventricular hypertrophy common

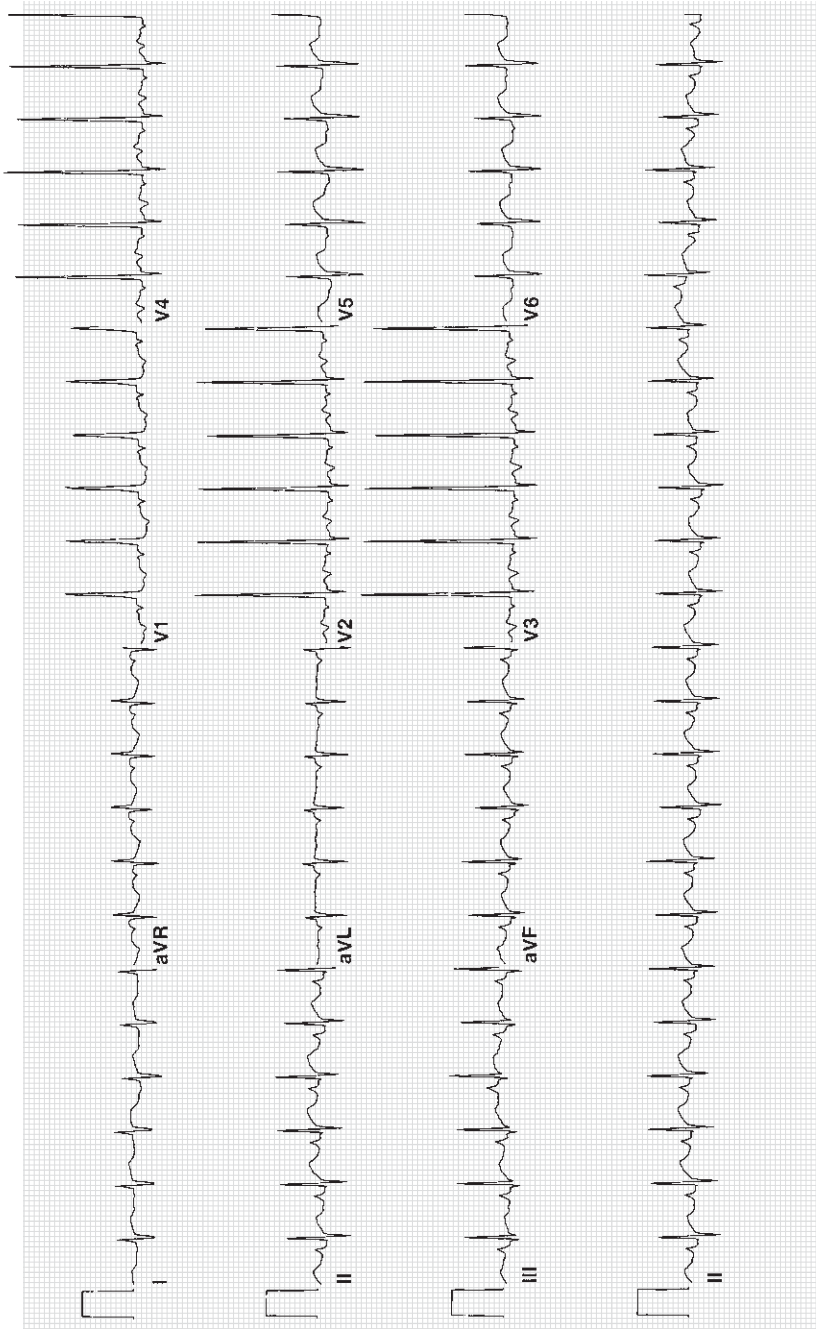


Figure 44 3-week-old infant with marked cyanosis.

Figure 44

- Sinus rhythm, 144 bpm
- QRS axis $+105^\circ$
- Right ventricular predominance:
 - R wave in V1 = 14 mm
 - S wave in V6 = 8 mm
- Nonspecific ST wave abnormalities

d-Transposition of the great arteries

- Normal ECG initially in newborns
- QRS axis: normal or mild right axis deviation
- Right ventricular hypertrophy, mild to moderate
 - Persistently upright T waves in V1 after first week of life
- Associated defects (ventricular septal defect, pulmonary stenosis, pulmonary hypertension) determine other ECG abnormalities

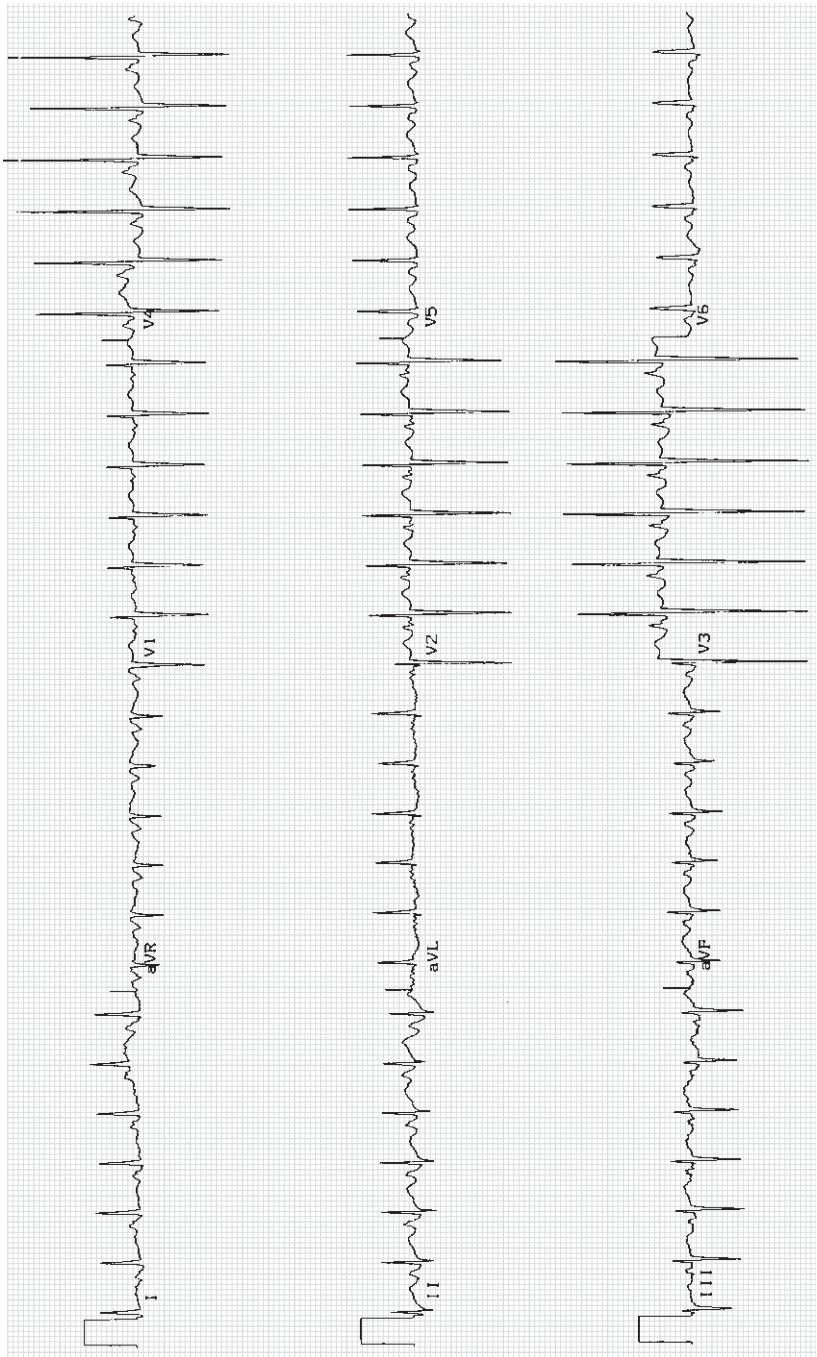


Figure 45 2-day-old infant with cyanosis.

Figure 45

- Sinus rhythm, 155 bpm
- Left axis deviation: QRS axis = -10°
- Right atrial enlargement
- Possible left atrial enlargement
- Left ventricular predominance:
 - Decreased R wave amplitude V1–V2
 - Prominent S wave in right precordium
- Nonspecific ST wave changes

Tricuspid atresia

- Variable QRS axis:
 - Normally related great arteries (Type I): Left axis deviation -30° to -120°
 - Transposed great arteries (Type II): QRS axis may be normal
- Biatrial enlargement in 80%
- Prolonged PR interval in $<15\%$
- Left ventricular hypertrophy in 85%, often with strain pattern
- Atrial arrhythmias increase with age

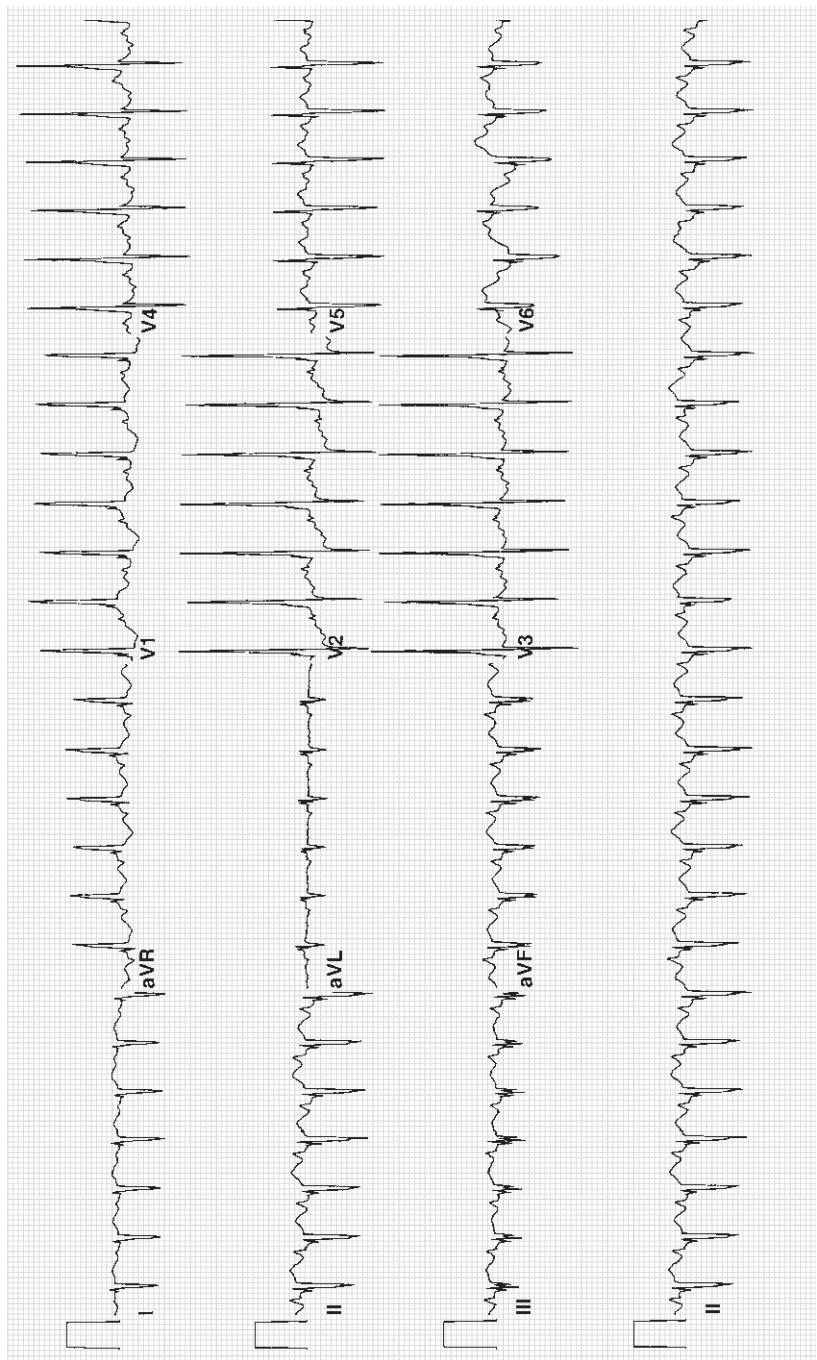


Figure 46 1-week-old male with tachypnea and mild cyanosis.

Figure 46

- Sinus rhythm, 160 bpm
- Right axis deviation: QRS axis $+220^\circ$
- Right ventricular predominance:
 rR' in V1, R' wave = 16 mm
 S wave in V6 = 9 mm

Total anomalous pulmonary venous connection

- Right axis deviation
- Right atrial enlargement
- Right ventricular hypertrophy (rsR' or rR' may be seen in lead V1)

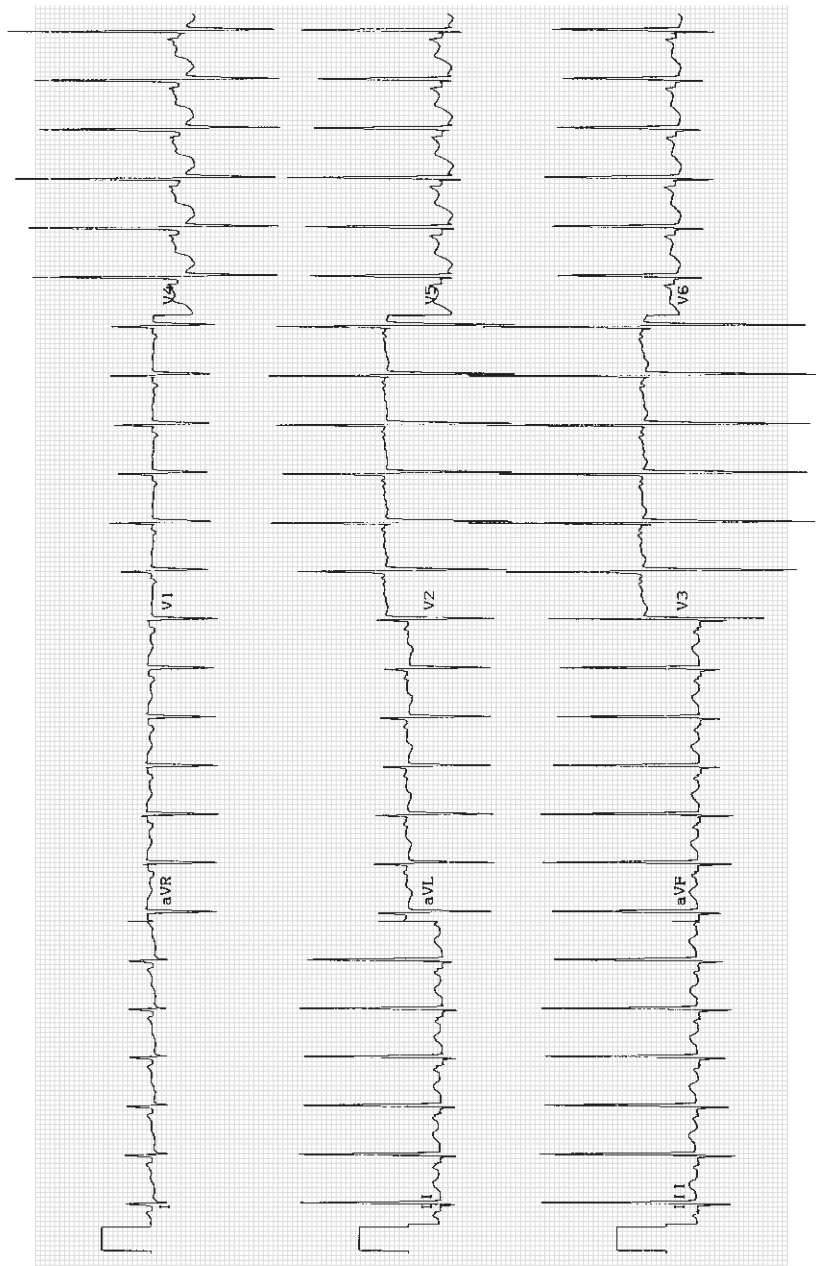


Figure 47 1-month-old infant with tachypnea, murmur, and cardiomegaly.

Figure 47

- Sinus rhythm, 148 bpm
- Possible left atrial enlargement
- Flattened T wave in V1 consistent with right ventricular hypertrophy
- Left ventricular hypertrophy with strain:
 - R wave in V6 = 24 mm
 - R wave in lead III = 30 mm
 - Q in III and V6 >6 mm
 - ST depression and T wave inversion in V4–V6

Truncus arteriosus

- QRS axis normal to mildly rightward ($+90^\circ$ to $+135^\circ$)
- Left atrial enlargement with increased pulmonary blood flow
- Left ventricular hypertrophy in 80%
- Combined ventricular hypertrophy in 70%

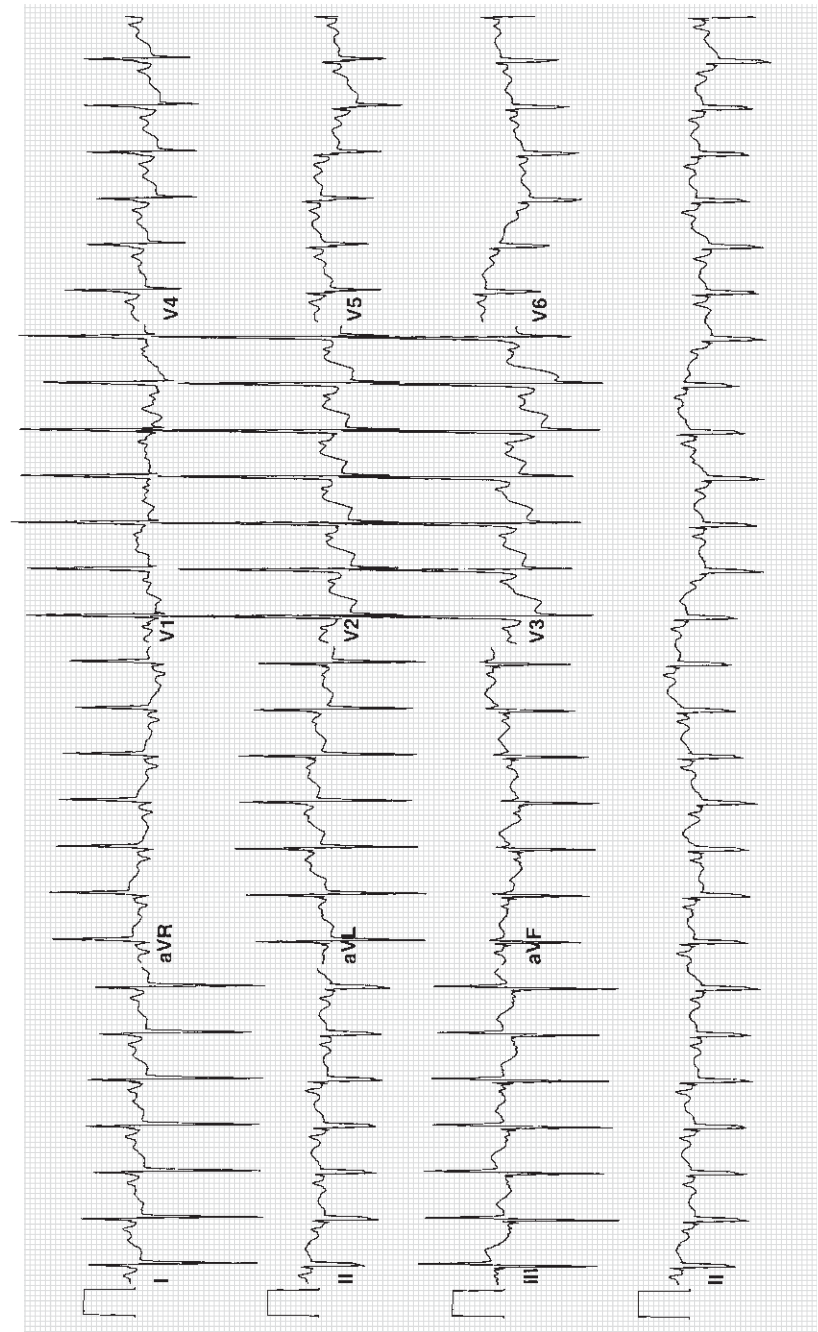


Figure 48 1-day-old infant with tachypnea, poor femoral pulses, and cardiomegaly.

Figure 48

- Sinus tachycardia, 166 bpm
- Right axis deviation: QRS +215°
- Right atrial enlargement
- Right ventricular hypertrophy:
 - Small Q in V1
 - R wave in V1 = 24 mm
 - S wave in V6 = 10 mm
- Decreased R wave amplitude in V5–V6
- Nonspecific T wave abnormality

Hypoplastic left heart syndrome

- ECG findings may be nonspecific, and may be normal in newborns
- QRS axis: right axis deviation
- Right atrial enlargement
- Left atrial enlargement with increased pulmonary blood flow
- Right ventricular hypertrophy
- Decreased left ventricular voltage:
 - Absent Q wave in V6
 - Poor R wave progression V4–V6
 - Small r wave in V6

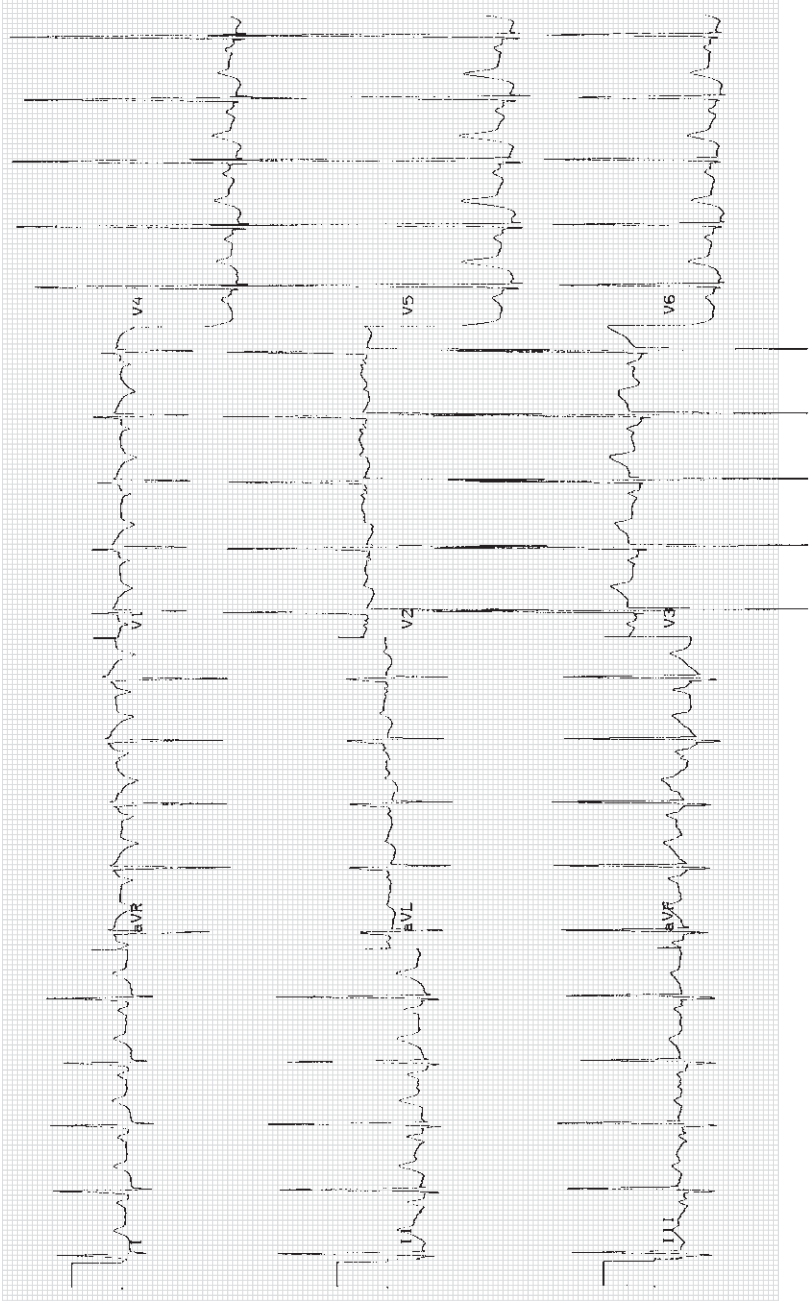


Figure 49 6-month-old infant with loud systolic murmur at right upper sternal border.

Figure 49

- Sinus rhythm, 116 bpm
- Left atrial enlargement
- Severe left ventricular hypertrophy:
R wave in V6 = 32 mm
ST depression in V4–V6

Aortic stenosis

- Normal ECG in 20%
- Left ventricular hypertrophy of varying degrees; may not correlate with degree of stenosis
- Severe stenosis: left ventricular hypertrophy with strain pattern:
ST depression, T wave inversion in left precordial leads
QRS–T wave angle discordance ($\geq 90^\circ$) reflects strain pattern

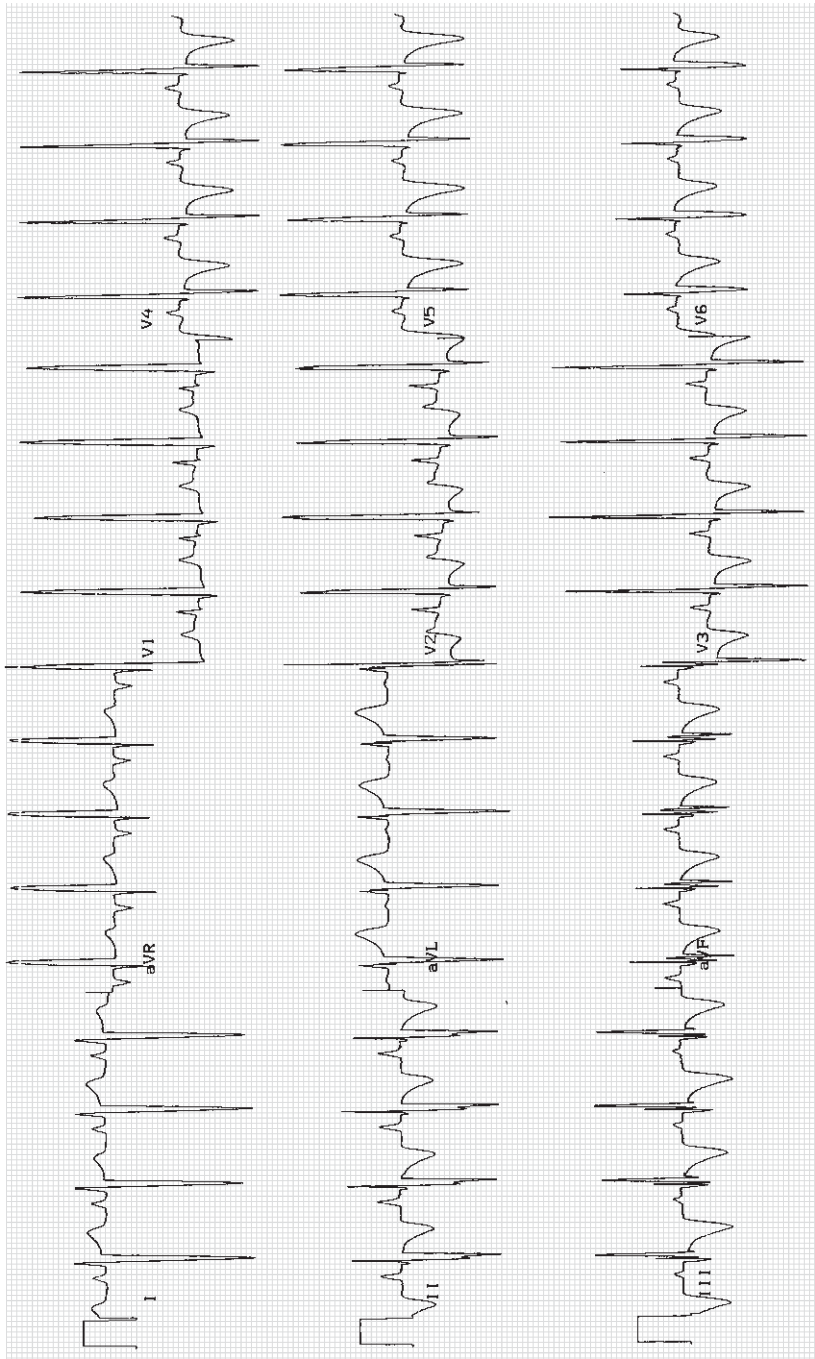


Figure 50 8-month-old infant with harsh systolic murmur at left upper sternal border.

Figure 50

- Normal sinus rhythm, 105 bpm
- Right axis deviation: QRS +180°
- Right atrial enlargement:
 - Tall peaked P waves in leads II, V1–V3
- Severe right ventricular hypertrophy with strain:
 - Q wave in V1
 - R wave in V1 = 31 mm
 - S wave in V6 = 13 mm
 - Upright T wave in V1
 - T wave inversion in leads II, III, aVF, V2–V6, with wide QRS–T angle discordance

Pulmonary stenosis

- Mild pulmonic stenosis:
 - Normal ECG in 30–40% rSR' or rR' in V1
- Moderate–severe pulmonic stenosis:
 - Right axis deviation: +90° to +210°
 - Right atrial enlargement
 - Right ventricular hypertrophy, reflects degree of stenosis

References

- Cayler GG, Ongley P, Nadas AS. Relation of systolic pressure in the right ventricle to the electrocardiogram. *N Engl J Med* 1958;258:979–982.
- Liebman J, Plonsey R. Electrocardiography. In Moss AJ, Adams FH, Emmanouilides CG, eds, *Heart Disease in Infants, Children and Adolescents*. Baltimore: Williams & Wilkins, 1977, 18–61.

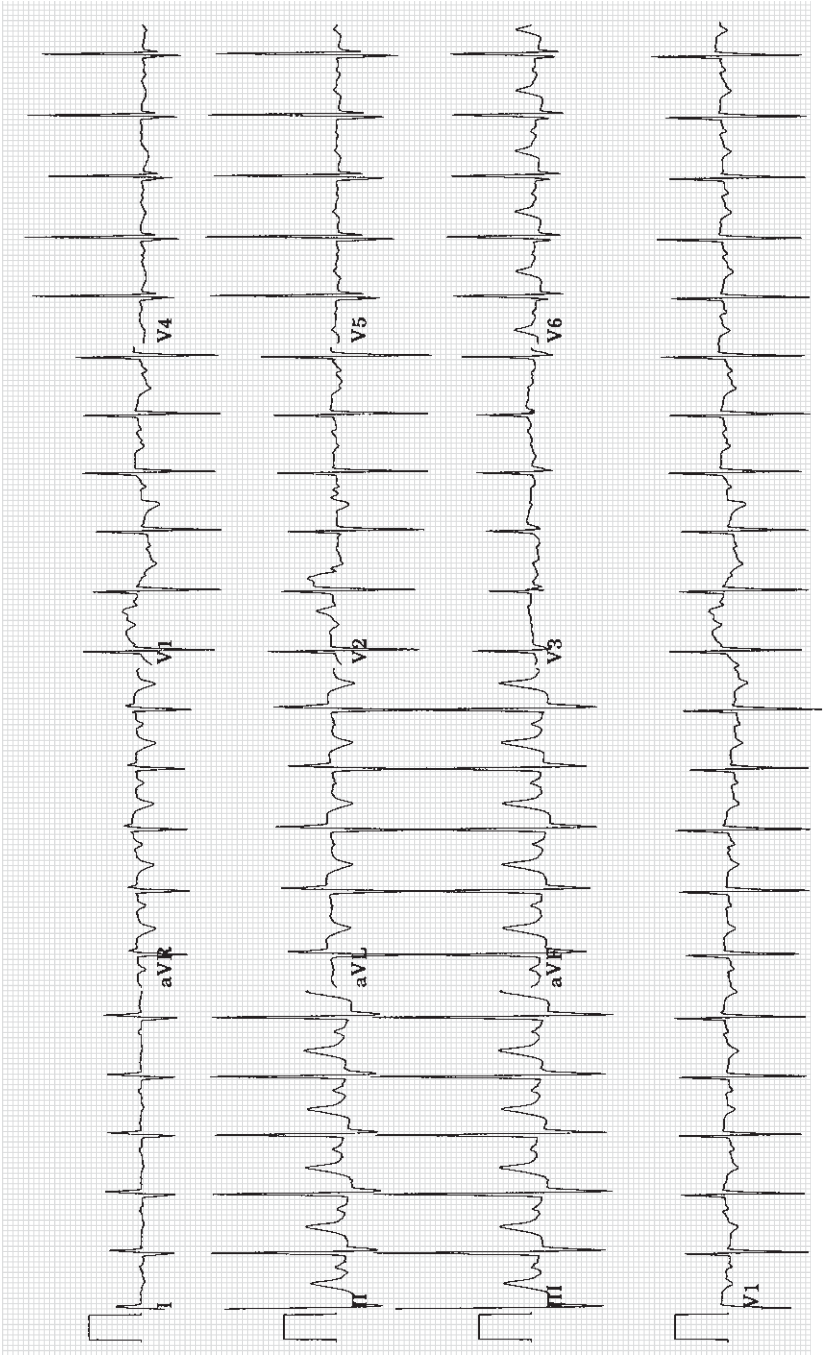


Figure 51 4-month-old baby with wheezing and cardiomegaly. (ECG courtesy of Richard Friedman, M.D.)

Figure 51

- Sinus rhythm, 130 bpm
- Pathological Q waves:
 Deep Q waves in I, aVL, V4–V6
 Q wave width in aVL = 0.04 sec
 ST segment depression in leads II, III, aVF, V6
- Left ventricular hypertrophy:
 R wave in III = 35 mm
- Pathological Q waves:
 Deep Q waves in I, aVL, V4–V6
 Q wave width in aVL = 0.04 sec

Anomalous origin of left coronary artery

- Normal QRS axis, occasional left axis deviation
- Deep Q waves and T wave inversion in I, aVL, V4–V6 indicate myocardial infarction:
 Deep Q wave >3 mm
 Wide Q wave >0.03 sec
- Left ventricular hypertrophy:
 Deep S wave in right precordium
 Tall R wave in leads V4–V6 with T wave changes
- Phases of myocardial infarction (anterior or anterolateral) may be evident

Reference

Johnsrude CL, Perry JC, Cecchin F, Smith EO, Fraley K, Friedman RA, Towbin JA. Differentiating anomalous left main coronary artery originating from the pulmonary artery in infants from myocarditis and dilated cardiomyopathy by electrocardiogram. *Am J Cardiol* 1995;**75**(1):71–74.

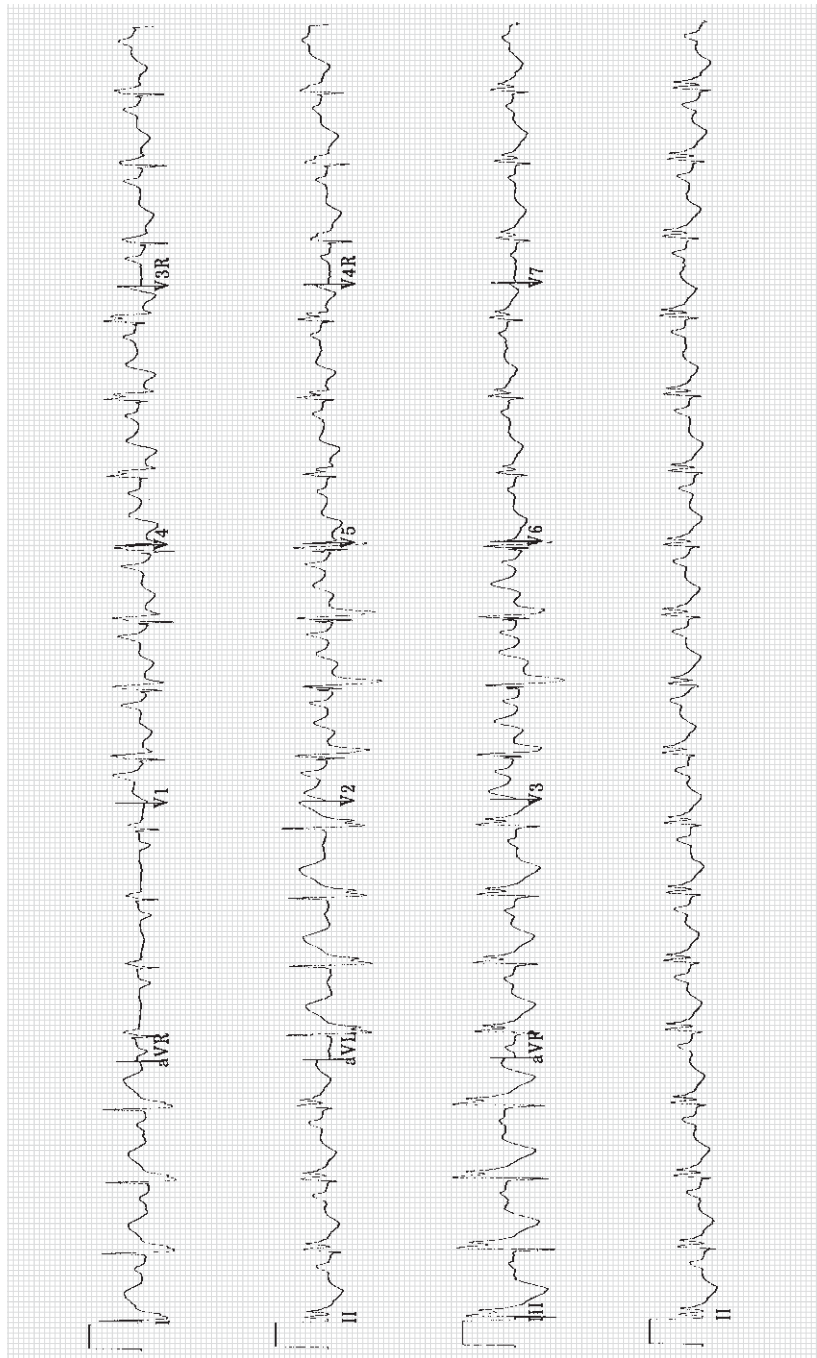


Figure 52 2-year-old boy with murmur, mild cardiomegaly, and frequent episodes of supraventricular tachycardia.

Figure 52

- Sinus rhythm, 110 bpm
- Right atrial enlargement
- Right bundle branch block:
QRS duration = 0.12 sec
rsR' in V1
- Prominent Q waves in III, aVF
- T wave inversion in inferolateral leads

Ebstein's anomaly of tricuspid valve

- QRS axis: rightward due to right bundle branch block, +90° to +150°
- Prolonged PR interval in 15–35%
- Right atrial enlargement in 60–95%
- Right bundle branch block in 75–80%:
Polyphasic QRS in V1–V3
Small amplitude QRS in V1–V3
- Pre-excitation pattern (Wolff–Parkinson–White syndrome) in 7–25%
- Supraventricular tachycardia (25–50%) increases with age; especially atrial flutter/fibrillation in older patients

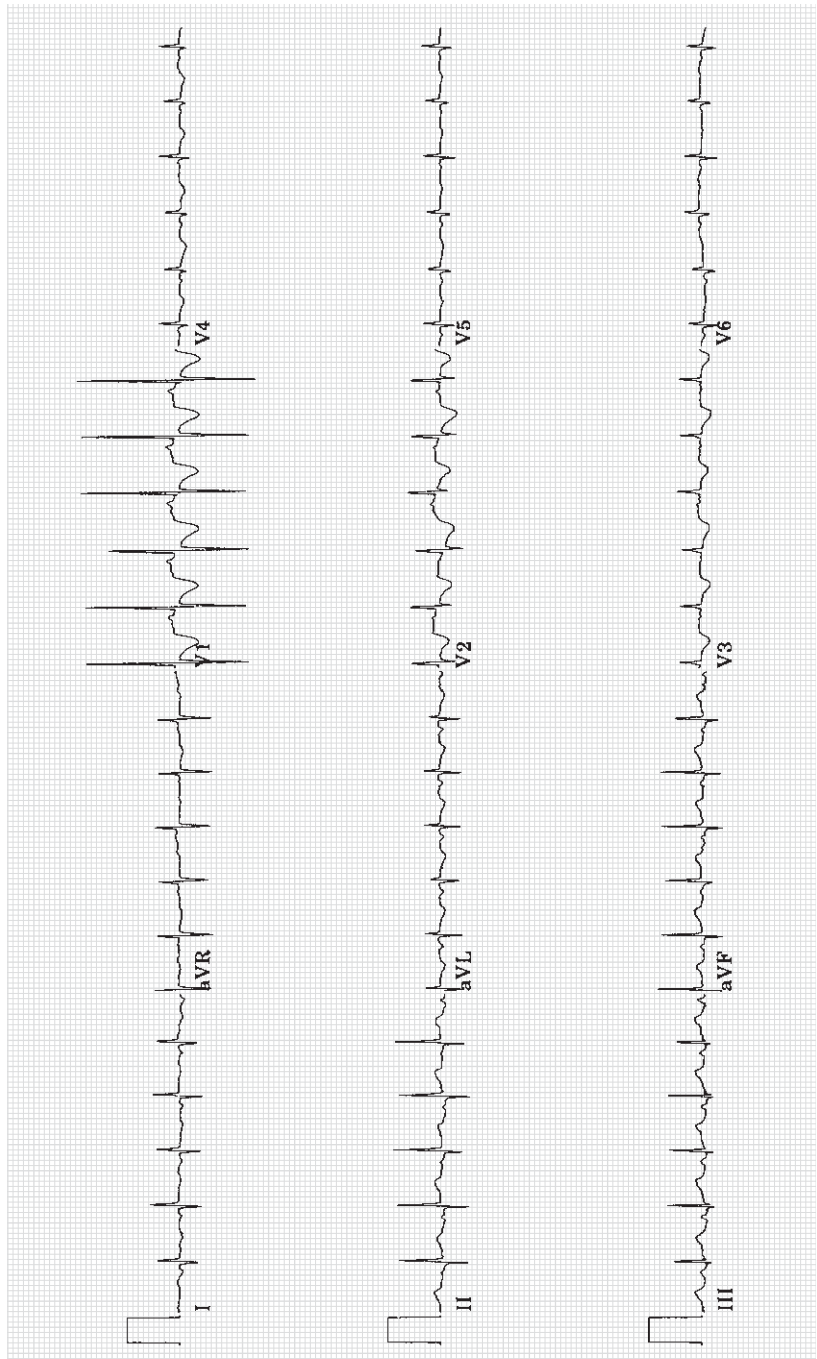


Figure 53 5-day-old infant with dextrocardia. (ECG courtesy of Richard Friedman, M.D.)

Figure 53

- Atrial rhythm, 140 bpm
- Left atrial rhythm: P wave inversion in I, aVL, V4–V6
- QR complexes and T wave inversion in leads I, aVL, V4–V6
- Decreased R wave amplitude in V4–V6

Dextrocardia

- Left atrial rhythm with atrial situs inversus implies “sinus rhythm”
- Tall right-sided QRS complexes reflect position of heart in thorax:
 - QRS voltage in V3R, V4R greater than in V3, V4
 - Decreasing QRS amplitude from V1 to V6

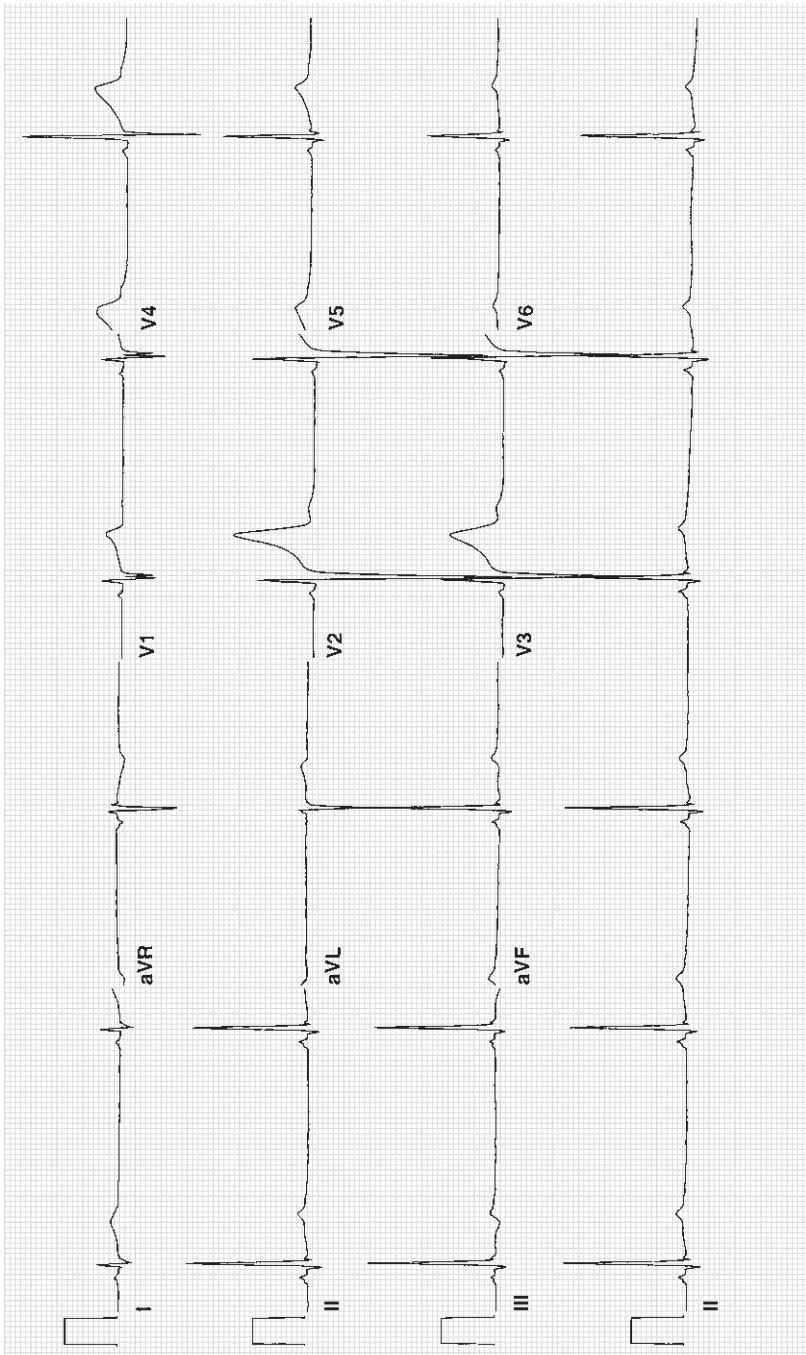


Figure 54 16-year-old male athlete.

Figure 54

- Sinus bradycardia, 34 bpm
- Upright P waves of normal morphology and axis in leads I, II, aVF
- Constant, normal PR interval with 1:1 atrioventricular conduction

Sinus bradycardia

- Slow sinus rate for age
- Criteria for bradycardia
Resting ECG:
HR <100 bpm, age 0–3 years
HR <60 bpm, age 3–9 years
HR <50 bpm, age >9 years
24-hour Holter criteria:
HR <80 bpm, 0–2 years
HR <60 bpm, 2–6 years
HR <45 bpm, 7–11 years
HR <40 bpm, >12 years
HR <30 bpm, athletes
- Commonly seen in athletes, including sinus pauses

References

- Friedman RA. Sinus and atrioventricular conduction disorders. In Deal BJ, Wolff GS, Gelband H, eds, *Current Concepts in Diagnosis and Management of Arrhythmias in Infants and Children*. Armonk NY: Futura Publishing, 1998, 89–90.
- Kugler JD. Sinus node dysfunction. In Gillette PC, Garson A Jr, eds, *Pediatric Arrhythmias: Electrophysiology and Pacing*. Philadelphia: WB Saunders, 1990, 250–300.

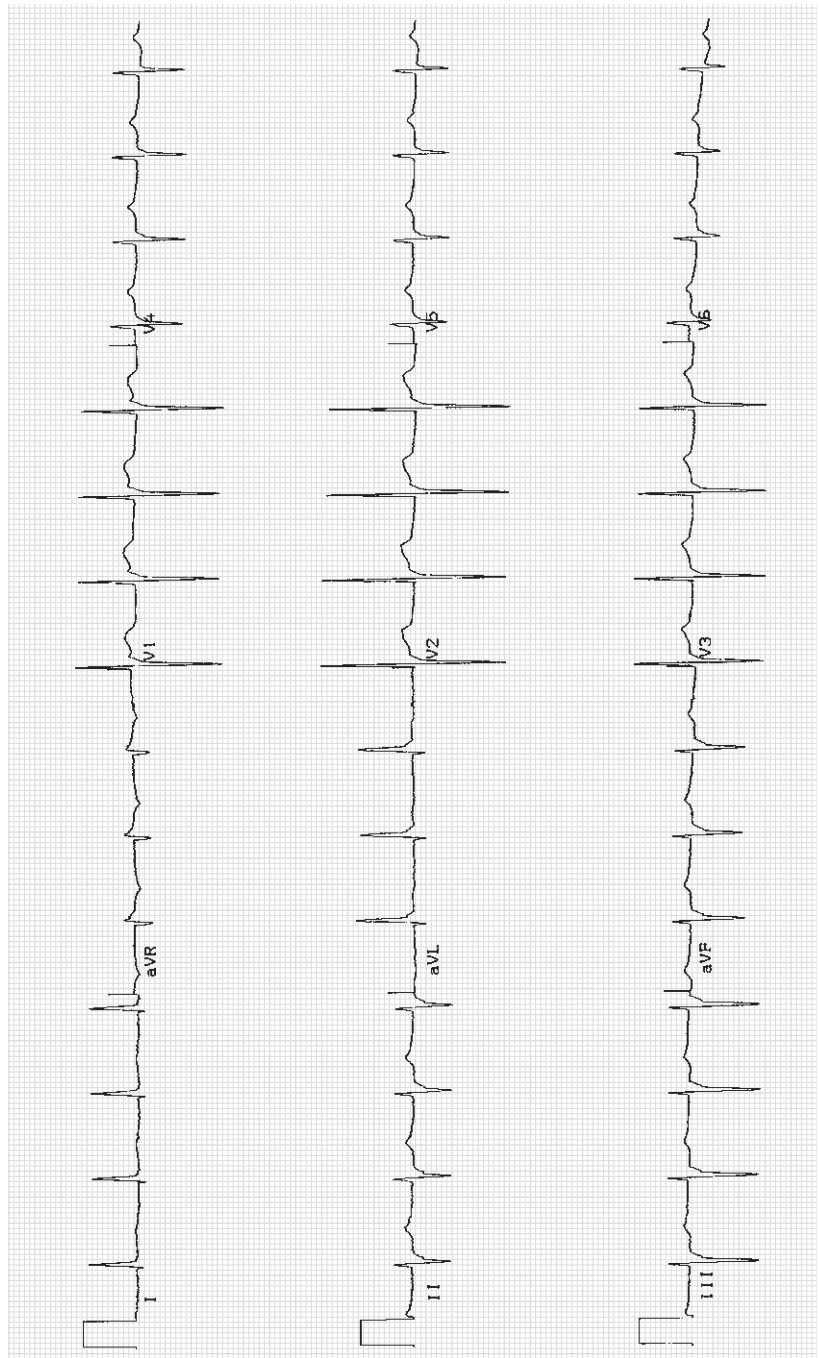


Figure 55 16-year-old female receiving sotalol for supraventricular tachycardia.

Figure 55

- Junctional rhythm, 90 bpm
- 1:1 ventriculoatrial conduction (Retrograde P waves seen as early notch on ST segment in V1)
- Left axis deviation: QRS axis -50°
- Upright T wave in V1: normal adult pattern
- Nonspecific T wave abnormality

Junctional rhythm

- Rates 40–100 bpm, vary with age
- Usually regular rhythm with narrow QRS (or QRS resembles patient's intrinsic QRS)
- P wave does not precede QRS complex; P waves may be dissociated or follow each QRS (retrograde conduction)
- Incidence: 13–45% of children on Holter monitor (transient)
- Consider:
 - Increased vagal tone (athletes, 20%)
 - Sinus node dysfunction
 - Postoperative congenital heart disease

Reference

Kugler JD. Benign arrhythmias: neonate throughout childhood. In Deal BJ, Wolff GS, Gelband H, eds, *Current Concepts in Diagnosis and Management of Arrhythmias in Infants and Children*. Armonk, NY: Futura Publishing, 1998, 73–75.

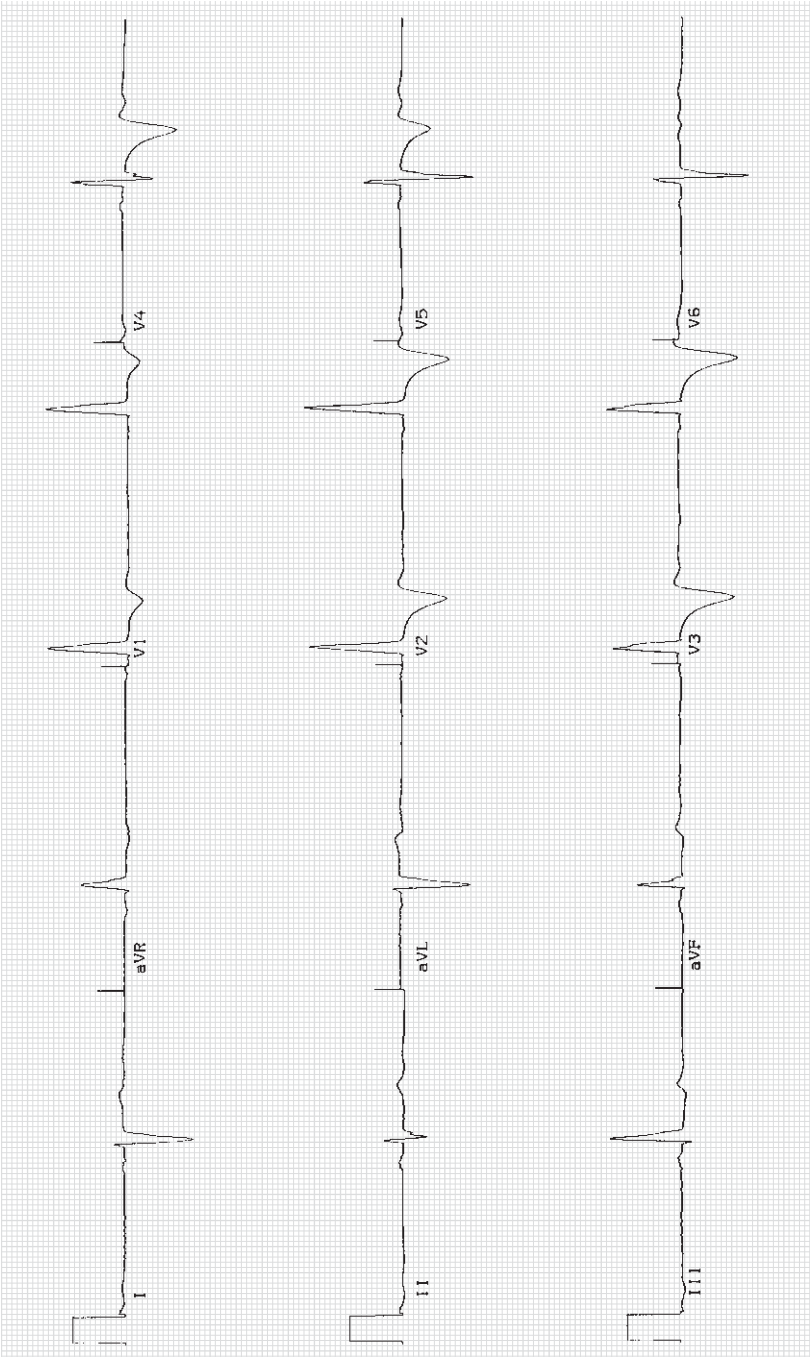


Figure 56 21-year-old male with transposition of the great arteries, status post-Mustard repair.

Figure 56

- Sinus bradycardia, 32 bpm, low amplitude P waves
- Right axis deviation: QRS axis $+150^\circ$
- Right ventricular hypertrophy with strain
- Decreased R wave amplitude in V5–V6

Sinus bradycardia: postoperative Mustard or Senning operation for transposition of the great arteries

- Sinus node dysfunction common after atrial switch operations; may be marked
- Right axis deviation, right ventricular hypertrophy due to systemic right ventricle
- Increased incidence of supraventricular tachycardia (atrial re-entry) with longer postoperative follow-up: 20–40%

Reference

Vetter VL, Tanner CS, Horowitz LN. Electrophysiologic consequences of the Mustard repair of d-transposition of the great arteries. *J Am Coll Cardiol* 1987;**10**(6):1265–1273.

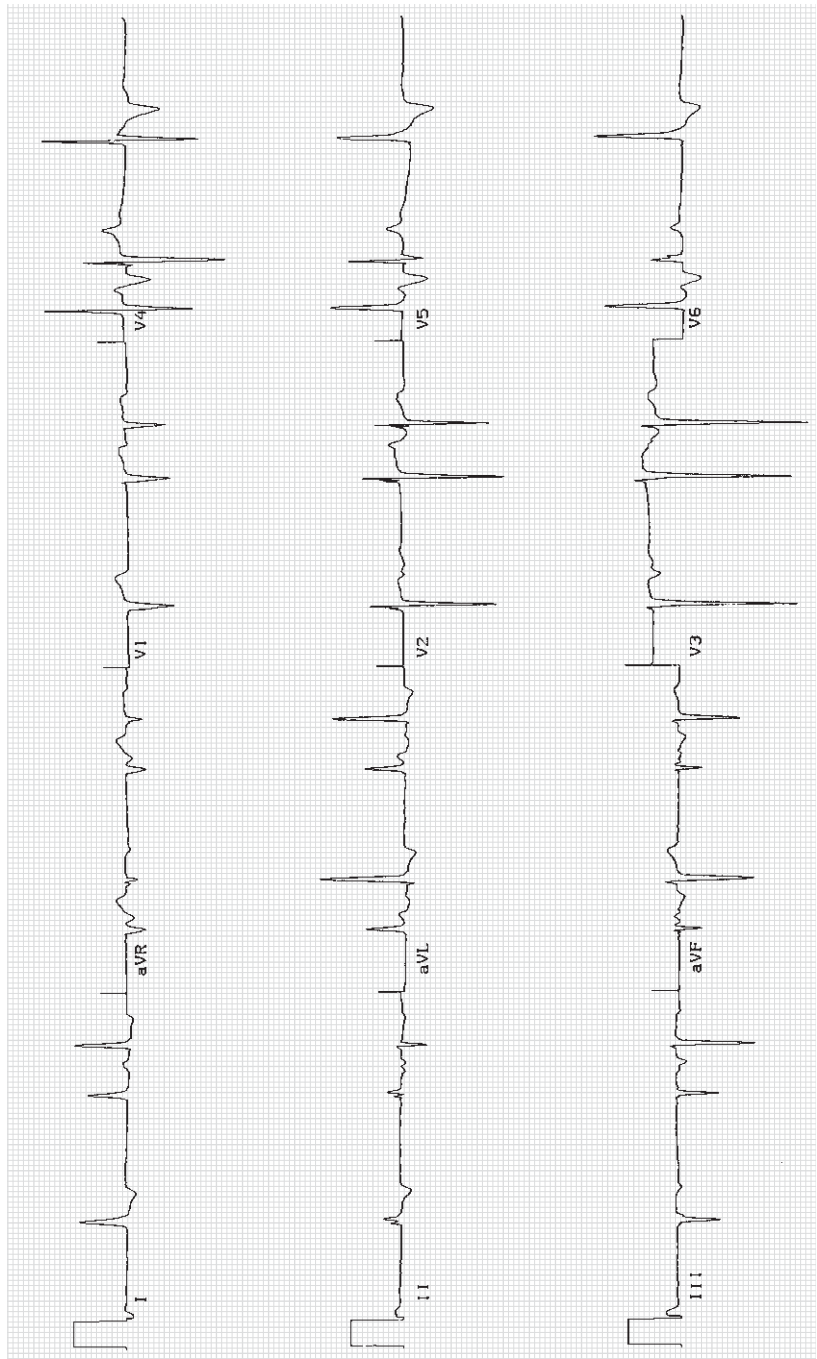


Figure 57 9-year-old girl, status post-Fontan procedure for tricuspid atresia.

Figure 57

- Sinus bradycardia with junctional escape rhythm followed by sinus capture beats: “escape–capture bigeminy”:
 Sinus P waves occur in ST segment of preceding junctional escape beats
 Sinus beats are occasionally conducted aberrantly
- Left axis deviation: QRS axis -25°
- Decreased R wave amplitude in V1–V3
- Left ventricular predominance with strain

Sinus bradycardia: postoperative Fontan procedure for single ventricle

- Sinus node dysfunction common after Fontan surgery; may be less marked than postoperative Mustard, Senning patients
- High incidence of supraventricular tachycardia (atrial re-entry): 40–60%
- Note: Sinus capture beats appear to be premature atrial contractions, but are not premature given underlying sinus bradycardia

Reference

Kavey REW, Gaum WE, Byrum CJ, Smith FC, Kveselis DA. Loss of sinus rhythm after total cavopulmonary connection. *Circulation* 1995;**92**(Suppl. II):II-304–II-308.

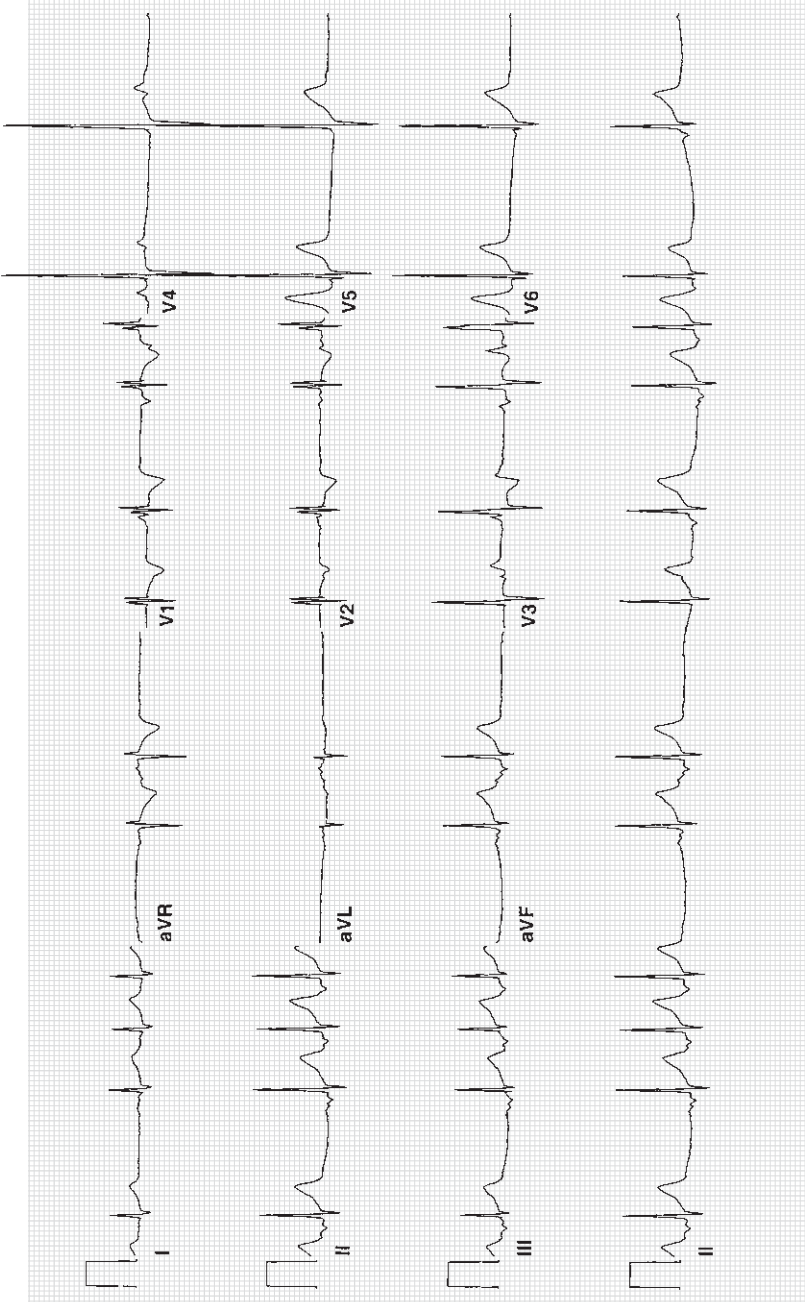


Figure 58 2-year-old girl referred for irregular heart rhythm.

Figure 58

- Irregular low right atrial rhythm with atrial bradycardia and junctional escape beats
- Low atrial premature atrial complexes, atrial couplets and triplets

Sick sinus syndrome

- Marked sinus bradycardia, sinus pauses or sinus arrest
- Sinus bradycardia alternating with sinus or atrial tachycardia
- Junctional or atrial escape beats during sinus pauses
- Also called tachycardia—bradycardia syndrome

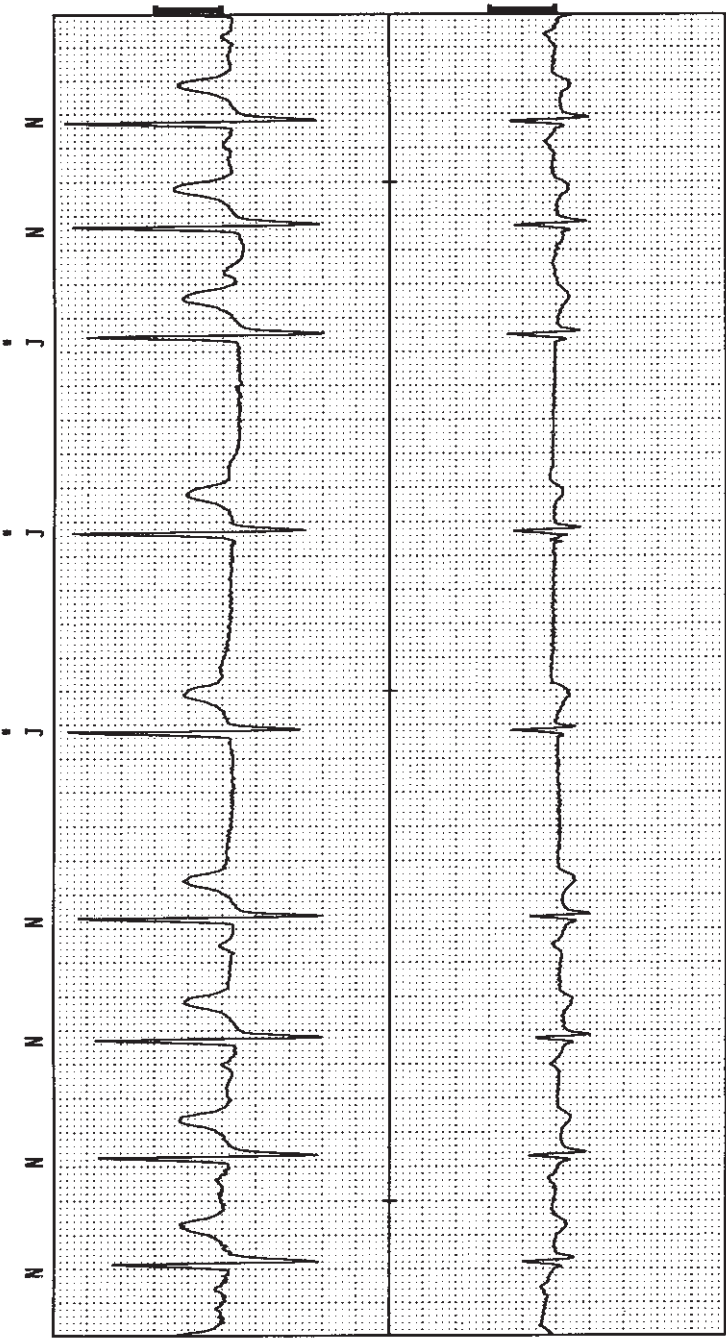


Figure 59 6-year-old boy referred for irregular heart rhythm.

Figure 59

- Sinus rhythm followed by sinus bradycardia, with junctional escape rhythm

Sick sinus syndrome

- Sinoatrial exit block:
 - Sinus impulses blocked in perinodal tissue
 - Bradycardia cycle length = multiple of underlying sinus cycle length
- Increasing incidence of atrial fibrillation with age
- Rare in children without structural heart disease

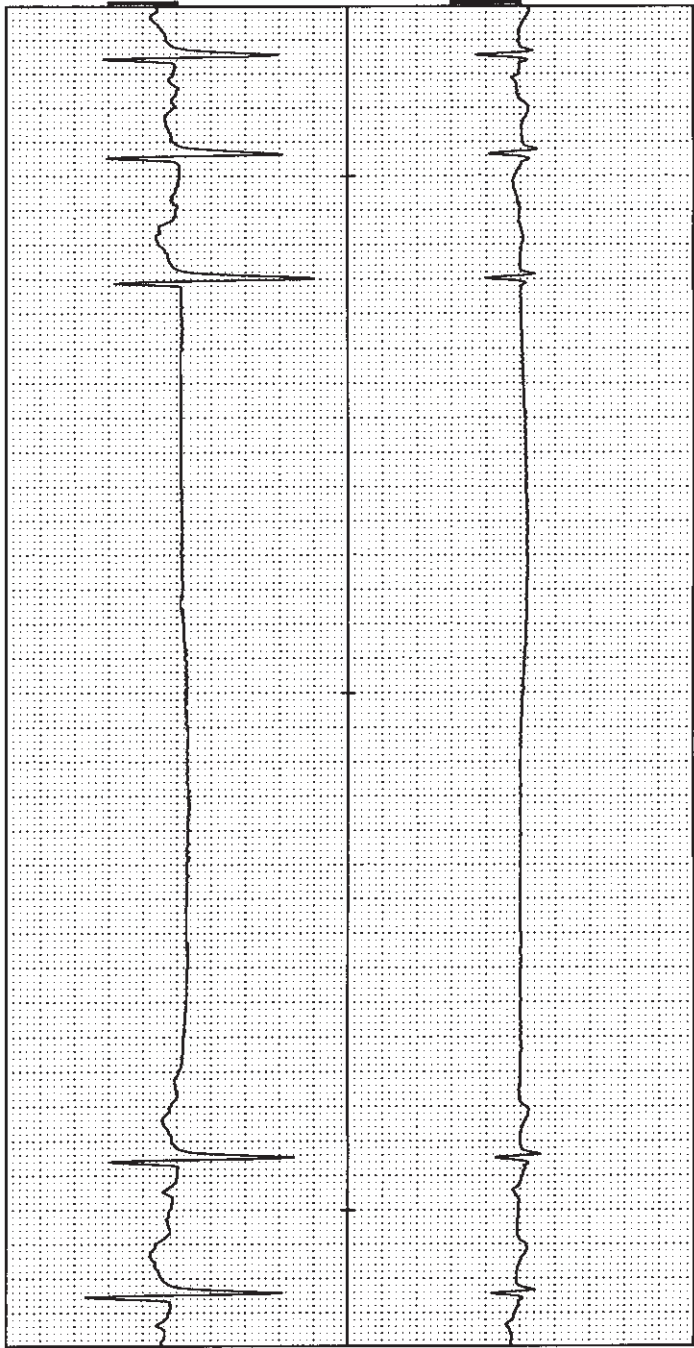


Figure 60 6-year-old boy referred for irregular heart rhythm.

Figure 60

- Sinus rhythm followed by profound sinus pause lasting 5 sec, with junctional escape beat

Sick sinus syndrome

- Sinoatrial exit block:
 Sinus impulses blocked in perinodal tissue
 Bradycardia cycle length = multiple of underlying sinus cycle length
- Increasing incidence of atrial fibrillation with age
- Rare in children without structural heart disease

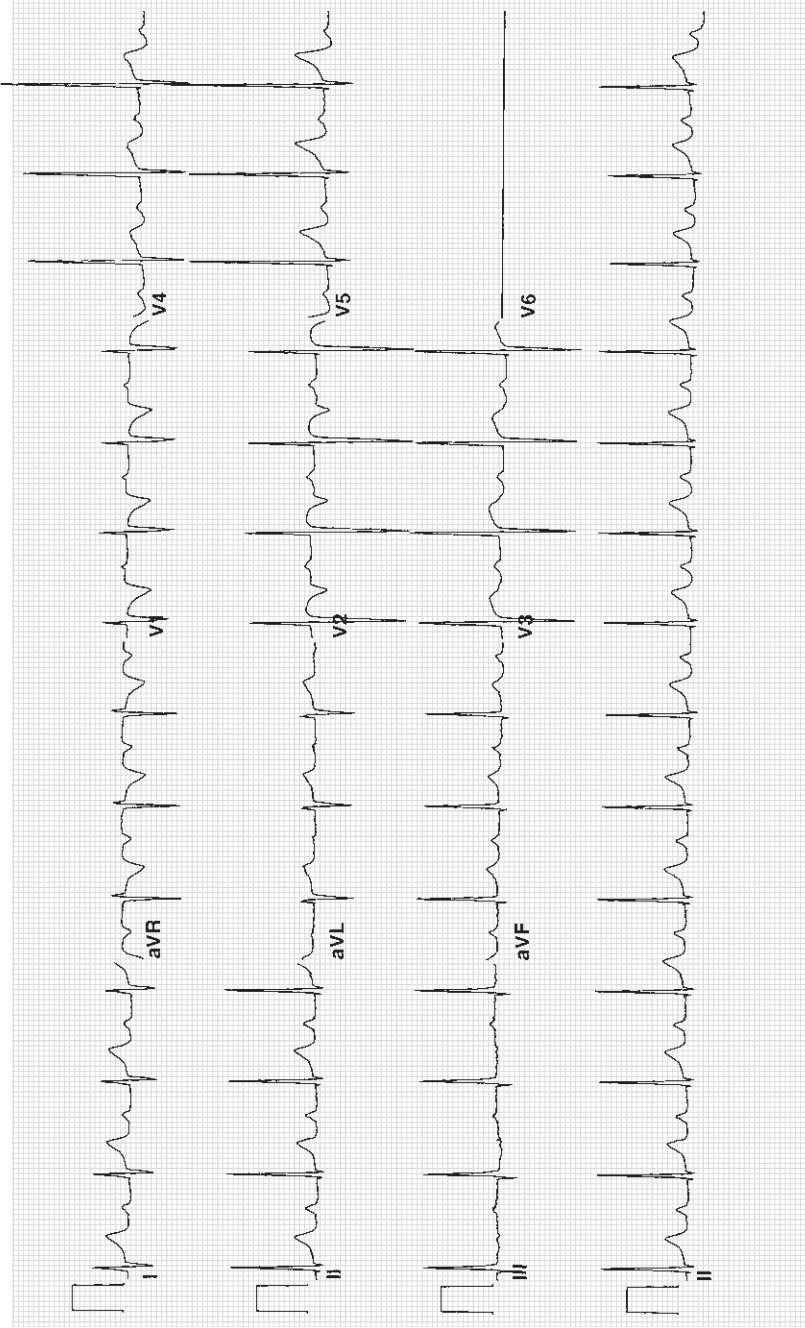


Figure 61 9-year-old boy with arthralgia and recent sore throat.

Figure 61

- Sinus rhythm, 84 bpm
- First degree atrioventricular block: prolonged PR interval = 0.28 sec
- Intact atrioventricular conduction

First degree atrioventricular (AV) block

- First degree atrioventricular block = PR interval >98% for age:
 - <1 year: PR >0.16 sec
 - 1–5 years: PR >0.18 sec
 - >5 years: PR >0.20 sec
- Prolonged PR interval reflects a delay rather than actual block.
- Acquired causes:
 - High vagal tone
 - Acute rheumatic fever
 - Hyper/hypo-kalemia, hypermagnesemia
 - Drug effect/toxicity (digoxin, other antiarrhythmic agents)
- Associated with some forms of congenital heart disease:
 - atrial septal defects (primum and secundum),
 - atrioventricular septal defect, double outlet right ventricle, Ebstein's anomaly of tricuspid valve, congenitally corrected transposition of the great arteries

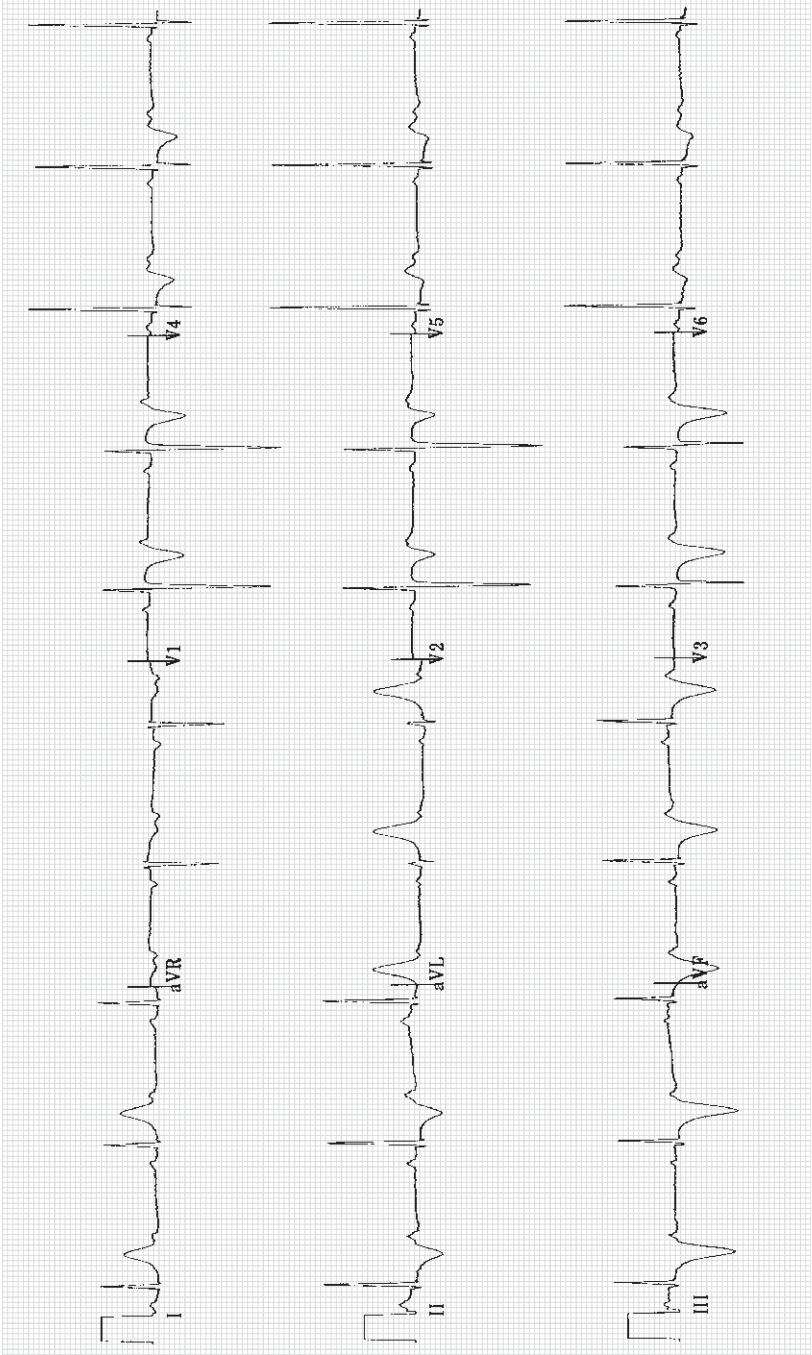


Figure 62 8-year-old boy with slow heart rate.

Figure 62

- Sinus rhythm, atrial rate 110 bpm
- 2:1 atrioventricular conduction, ventricular rate 55 bpm
- Borderline left ventricular hypertrophy
- Marked T wave inversion in inferolateral leads

Second degree atrioventricular (AV) block

- 2° atrioventricular block: some but not all P waves followed by QRS complex
- 3 types of 2° atrioventricular block:
 - Mobitz 1 (Wenckebach): Progressive PR lengthening prior to block
 - Mobitz II: No change in PR interval before block
 - 2:1 atrioventricular block: Every other P wave followed by QRS
- Advanced 2° atrioventricular block:
 - 2:1 AV block *or* multiple consecutive P waves not followed by QRS

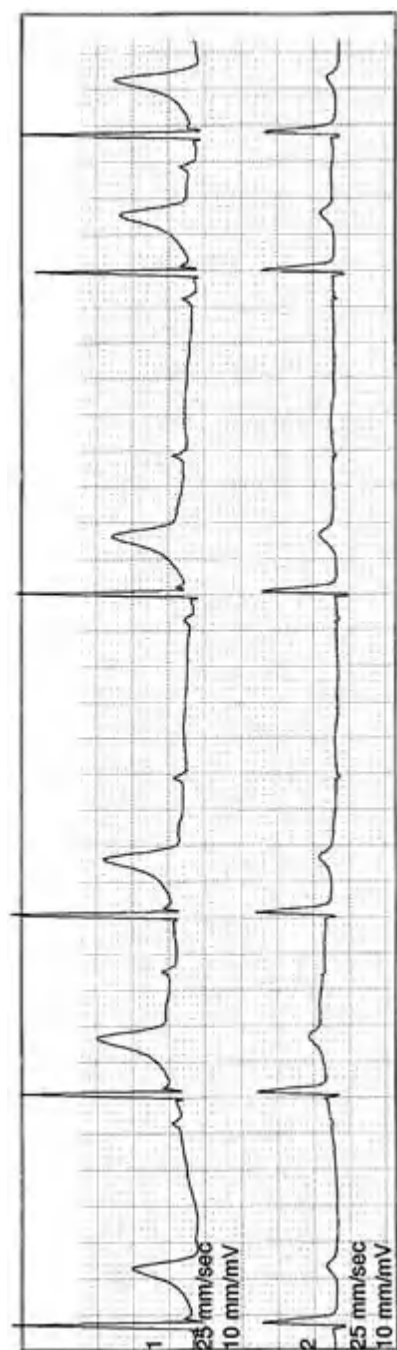


Figure 63 10-year-old boy with resolving myocarditis.

Figure 63

- Sinus rhythm with gradual prolongation of PR interval, followed by block

**Second degree atrioventricular block:
(Wenckebach, Mobitz I)**

- Progressive PR lengthening prior to atrioventricular block
- Normal finding during sleep
- Associated with increased vagal tone in athletes, drug effect, electrolyte abnormality, myocarditis

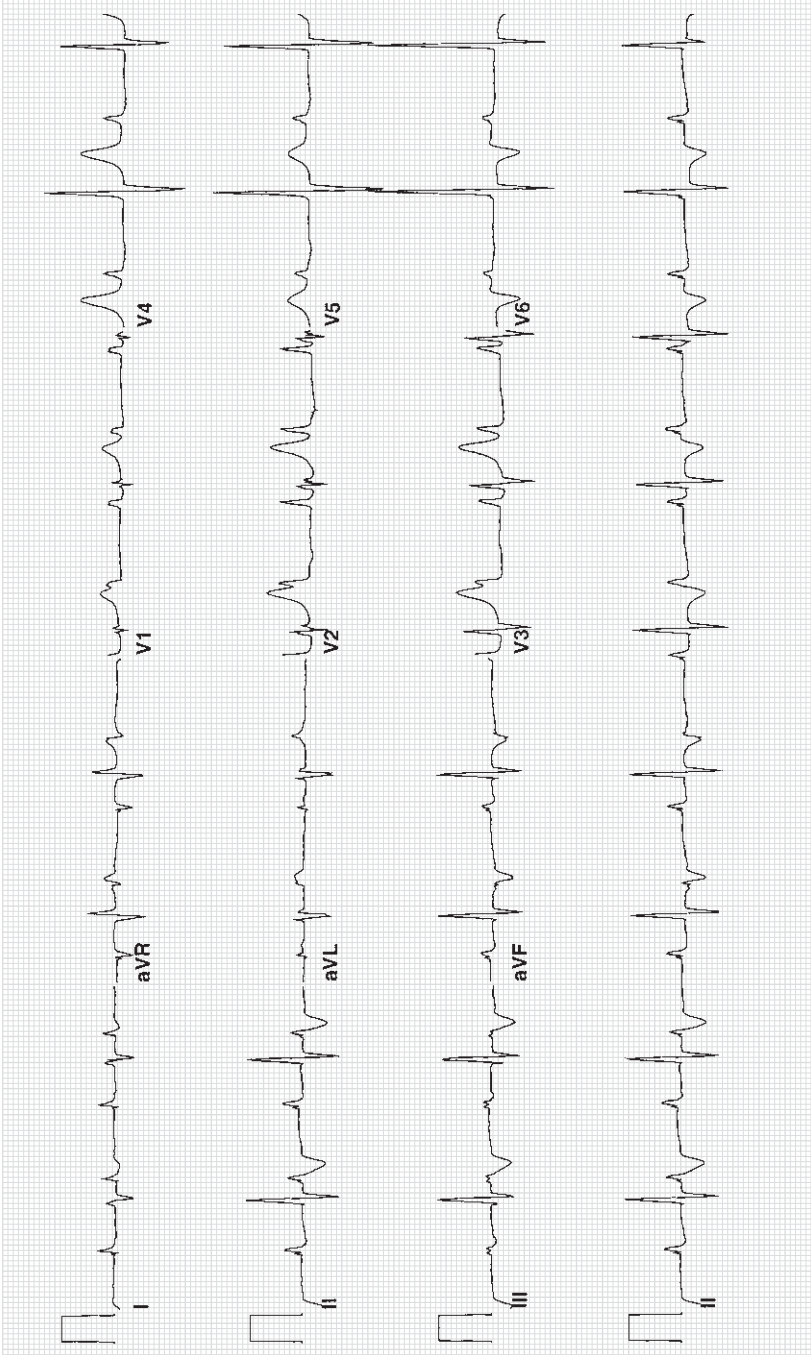


Figure 64 8-year-old girl with congenital complete heart block.

Figure 64

- Sinus rhythm, atrial rate 110 bpm
- Complete atrioventricular dissociation
- Regular junctional escape rhythm, 55 bpm
- Right atrial enlargement
- Right ventricular hypertrophy:
S wave in V6 = 8 mm
Upright T wave in V1
- T wave inversion in inferolateral leads: consider
cardiomyopathy, or post-chronic ventricular pacing
changes

Third degree atrioventricular block

- Complete dissociation between P waves and QRS complexes *and* atrial rate > ventricular rate
- Escape rhythm may be junctional (narrow QRS) or ventricular (wide QRS)
- Congenital 3° atrioventricular block:
Maternal autoimmune disorder
Long QT syndrome
Structural heart disease (asplenia syndrome,
congenitally corrected transposition of the great
arteries)

References

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- Michaelsson M, Jonzon A, Riesenfeld T. Isolated congenital complete atrioventricular block in adult life. A prospective study. *Circulation* 1995;**92**:442–449.

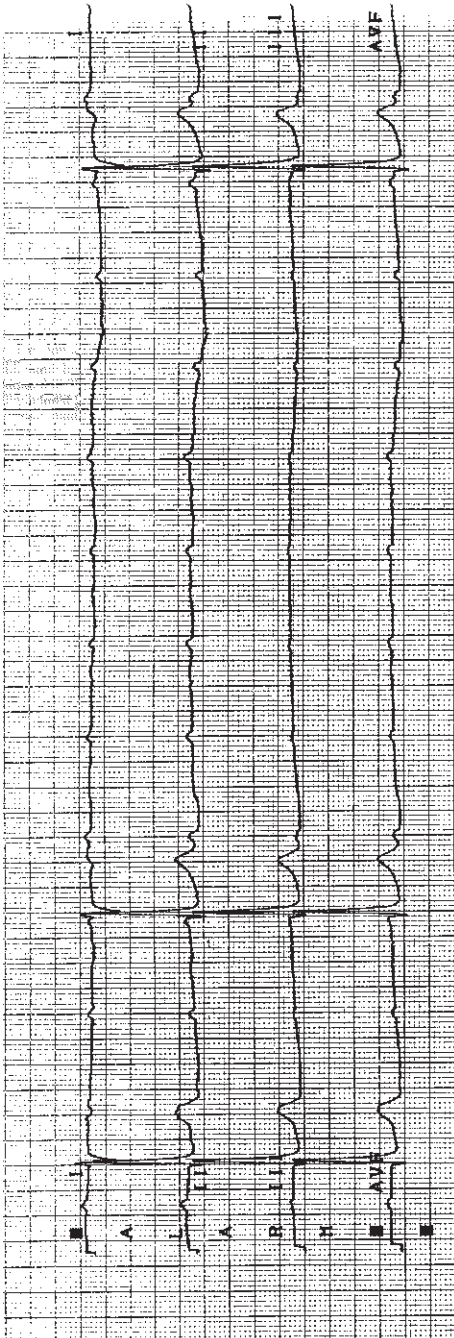


Figure 65 15-year-old girl with syncope during phlebotomy.

Figure 65

- Sinus rate, 80 bpm
- Third degree atrioventricular block:
Atrial rate, 80 bpm
Very slow junctional escape rhythm
- Junctional exit block:
2-sec interval between first two QRS complexes
6-sec interval between last two QRS complexes

Third degree atrioventricular block

- Complete atrioventricular block may be transient or permanent
- Acquired third degree atrioventricular block:
Postoperative repair of congenital heart disease involving ventricular septal defect closure, subaortic resection, aortic valve replacement
Occasionally seen in neurally mediated syncope: cardio-inhibition suppresses junctional and ventricular automaticity as well as AV conduction
Infections: myocarditis, Lyme disease
Drug induced, ingestion

Reference

Weindling SN, Saul JP, Mayer JE, Wessel D, Walsh EP. Duration of complete atrioventricular block after congenital heart disease surgery. *Am J Cardiol* 1998;**82**(4):525–527.

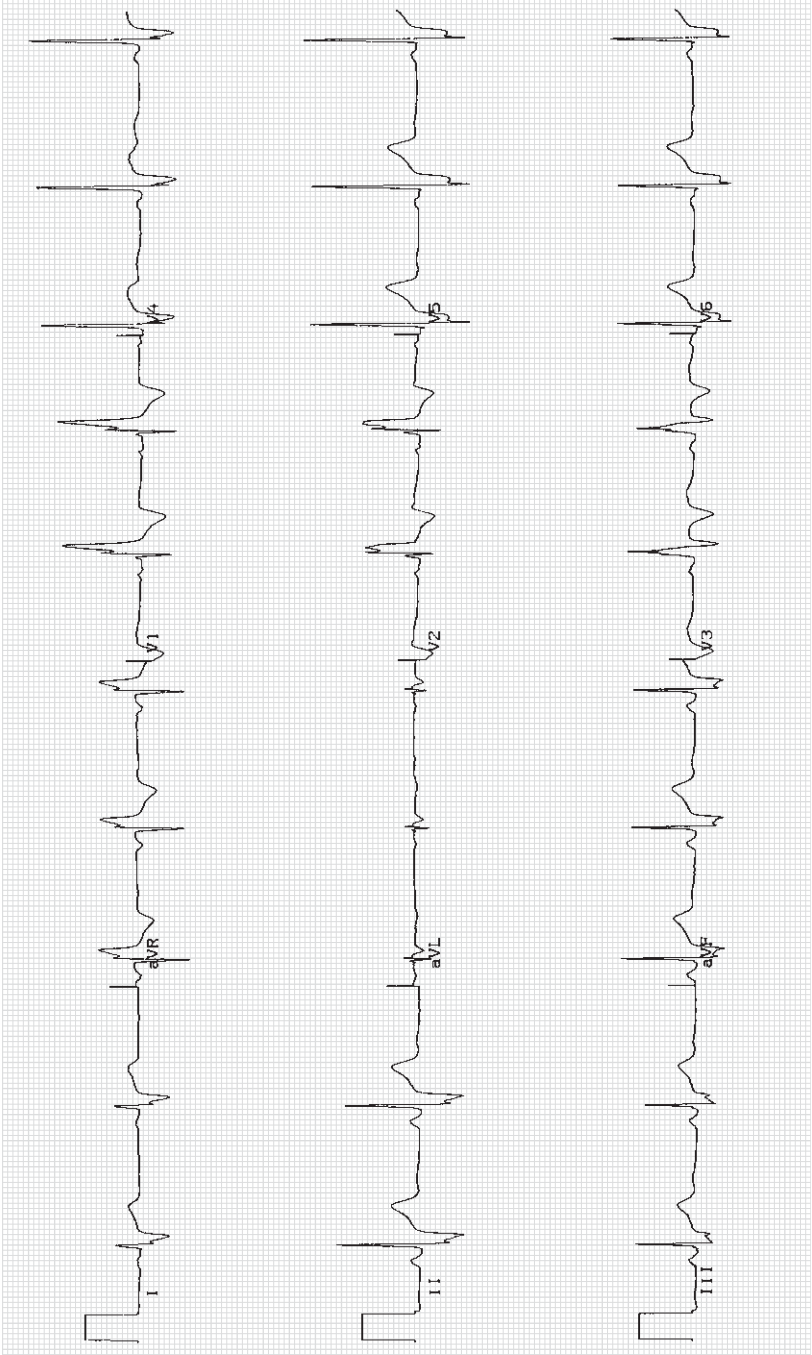


Figure 66 13-year-old boy, status post-repair of ventricular septal defect.

Figure 66

- Sinus bradycardia, 58 bpm
- Complete right bundle branch block:
QRS duration = 0.15 sec
rsR' pattern in V1, V2
Slurred, wide S wave in V6

Complete right bundle branch block (RBBB)

Criteria for RBBB:

- QRS duration prolonged for age:

<4 years	>0.09 sec
4–12 years	>–0.10 sec
>12 years	>0.12 sec
- Normal initial QRS forces
- Terminal conduction delay:

V1:	broad slurred R' wave (rSR') I,
V6:	Deep broad S wave
- Usually acquired, following intracardiac surgery
(ventricular septal defect closure, right ventriculotomy, cardiac transplant); carditis
- Rarely congenital (familial RBBB)

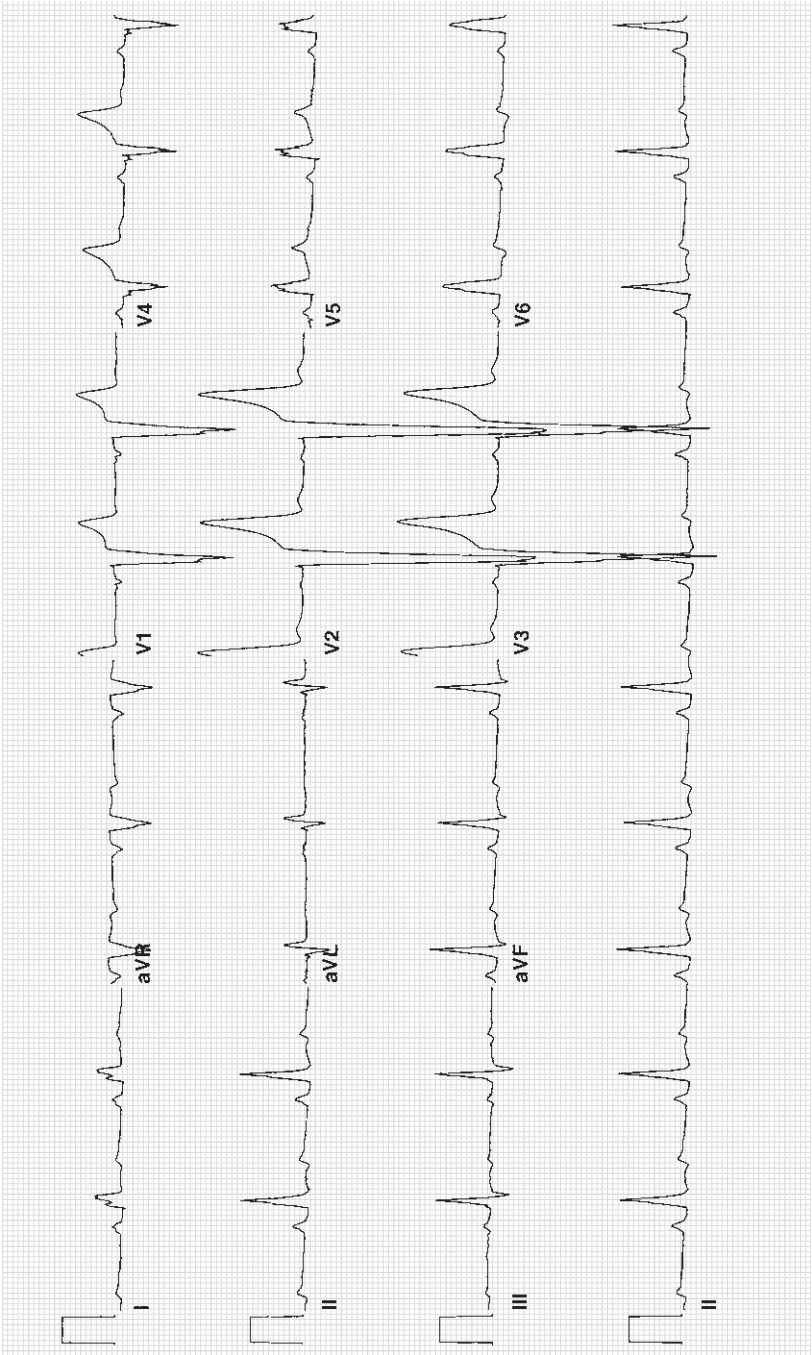


Figure 67 13-year-old boy with hypertrophic cardiomyopathy.

Figure 67

- Sinus rhythm, 60 bpm
 - Left atrial enlargement
 - Left bundle branch block
 - QRS duration = 0.135 sec
 - Wide slurred S wave in V1–V3
 - Slurred upstroke R wave in V5–V6
-
- Criteria for LBBB:
- Prolonged QRS duration for age:
 - <4 years >0.09 sec
 - 4–12 years >0.10 sec
 - >12 years >0.12 sec
 - Tall, broad, monophasic R waves in I, V5, and V6
 - No Q waves in I, V5, V6
 - Delayed initial QRS forces in V5 and V6
 - Usually QS or rS in V1
 - Rare in children
 - Usually acquired: postoperative subaortic or septal resection, carditis, cardiomyopathy, myocardial infarction

Complete left bundle branch block (LBBB)

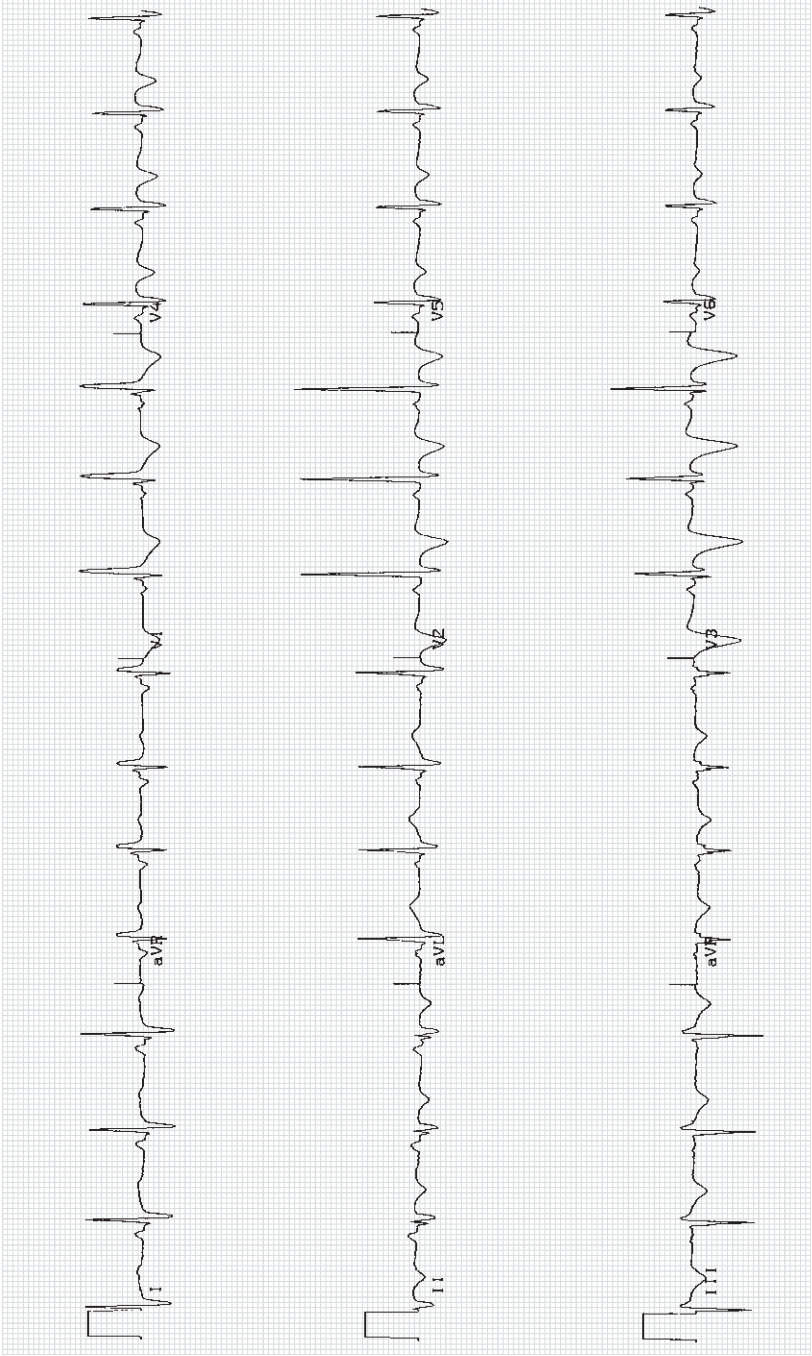


Figure 68 10-year-old boy with fever, tachypnea, and malaise.

Figure 68

- Sinus rhythm, 84 bpm
- Left axis deviation: QRS axis -45°
- Right bundle branch block:
QRS duration 0.12 sec
rsR' in V1
Slurred S wave in V6
- T wave inversion in inferolateral leads

Bifascicular block

- In children, usually consists of right bundle branch block and left anterior hemiblock (manifests as left axis deviation)
- Acquired:
Postoperative tetralogy of Fallot (25%)
Myocarditis, cardiomyopathy, heart transplantation
- Congenital:
Kearns–Sayre syndrome
Familial conduction disorders

Reference

Roberts NK, Perloff JK, Kark RAP. Cardiac conduction in the Kearns–Sayre syndrome (a neuromuscular disorder associated with progressive external ophthalmoplegia and pigmentary retinopathy). *Am J Cardiol* 1979;44:1396.

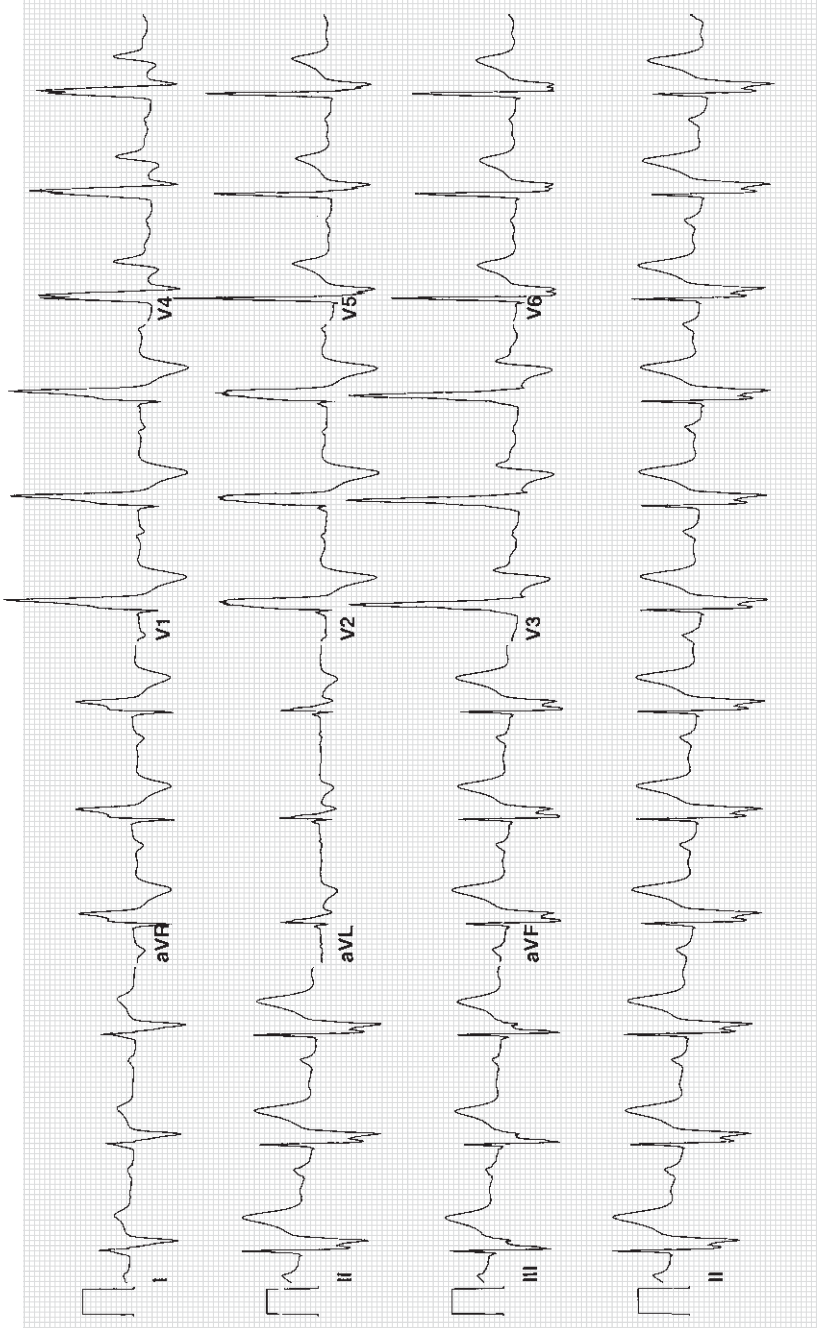


Figure 69 14-year-old boy, status post-repair of tetralogy of Fallot.

Figure 69

- Sinus rhythm, 73 bpm
- Right axis deviation: QRS axis $+240^\circ$
- First degree atrioventricular block: PR interval 0.22 sec
- Right bundle branch block:
 - QRS duration 0.16 sec
 - rsR' pattern in V1–V2
 - Slurred S wave in V5–V6

Trifascicular block

- Misnomer: more accurately described as “delay” rather than “block” because true trifascicular block results in complete atrioventricular block
- Criteria for trifascicular block:
 - Bifascicular block with 1° or 2° atrioventricular block
- Uncommon in children. Most commonly seen after surgery for tetralogy of Fallot, double outlet right ventricle, complete atrioventricular septal defect

154 Supraventricular Tachycardia

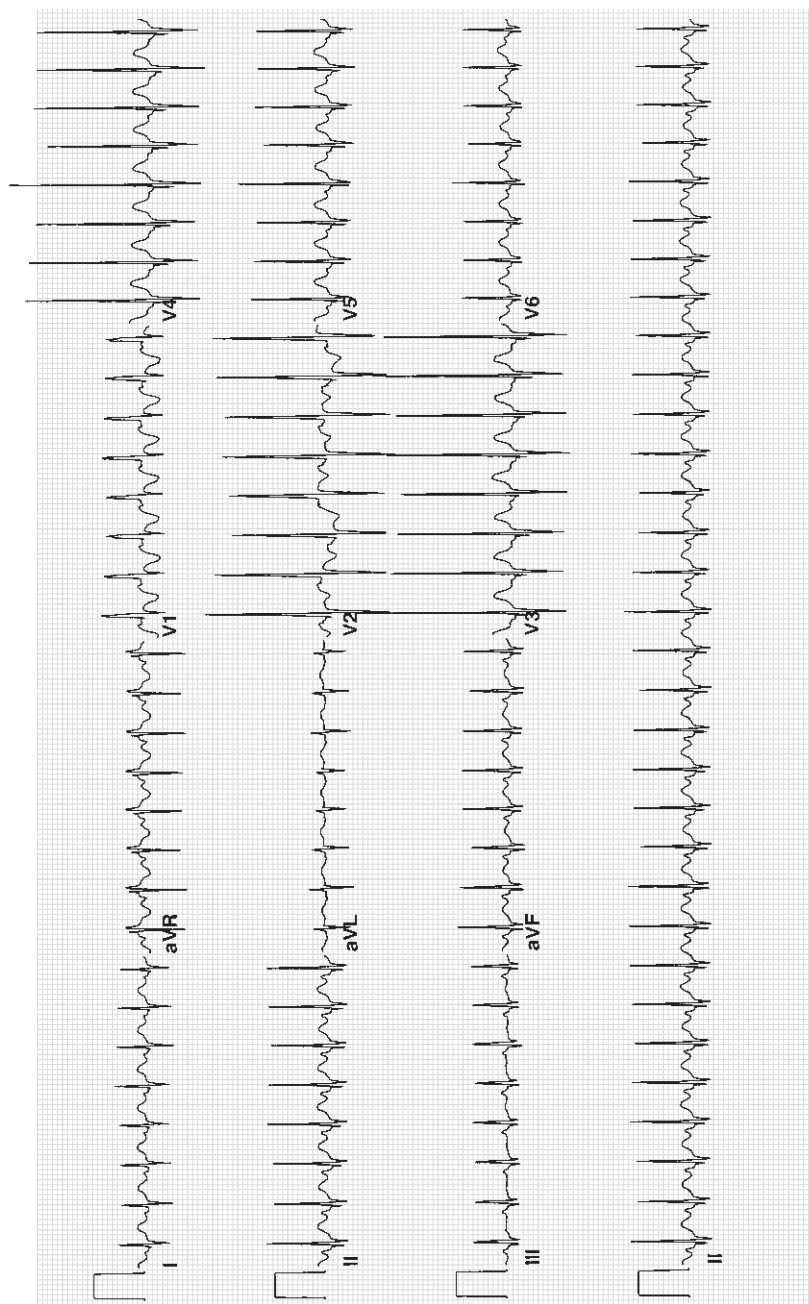


Figure 70 3-month-old infant with fever.

Figure 70

- Sinus tachycardia, 193 bpm
- Right ventricular predominance, normal for age

Sinus tachycardia

- Normal P wave precedes each QRS with normal PR interval
- Sinus tachycardia rates vary with age:

<1 year	>180 bpm
1–3 years	>150 bpm
>3 years	>140 bpm
>12 years	>120 bpm
- Associated with:
 - Crying, anxiety, pain, fever,
 - Hypovolemia, exertion, anemia
 - Drugs (bronchodilators, inotropic agents, pseudoephedrine, cocaine)
 - Heart failure, thyrotoxicosis

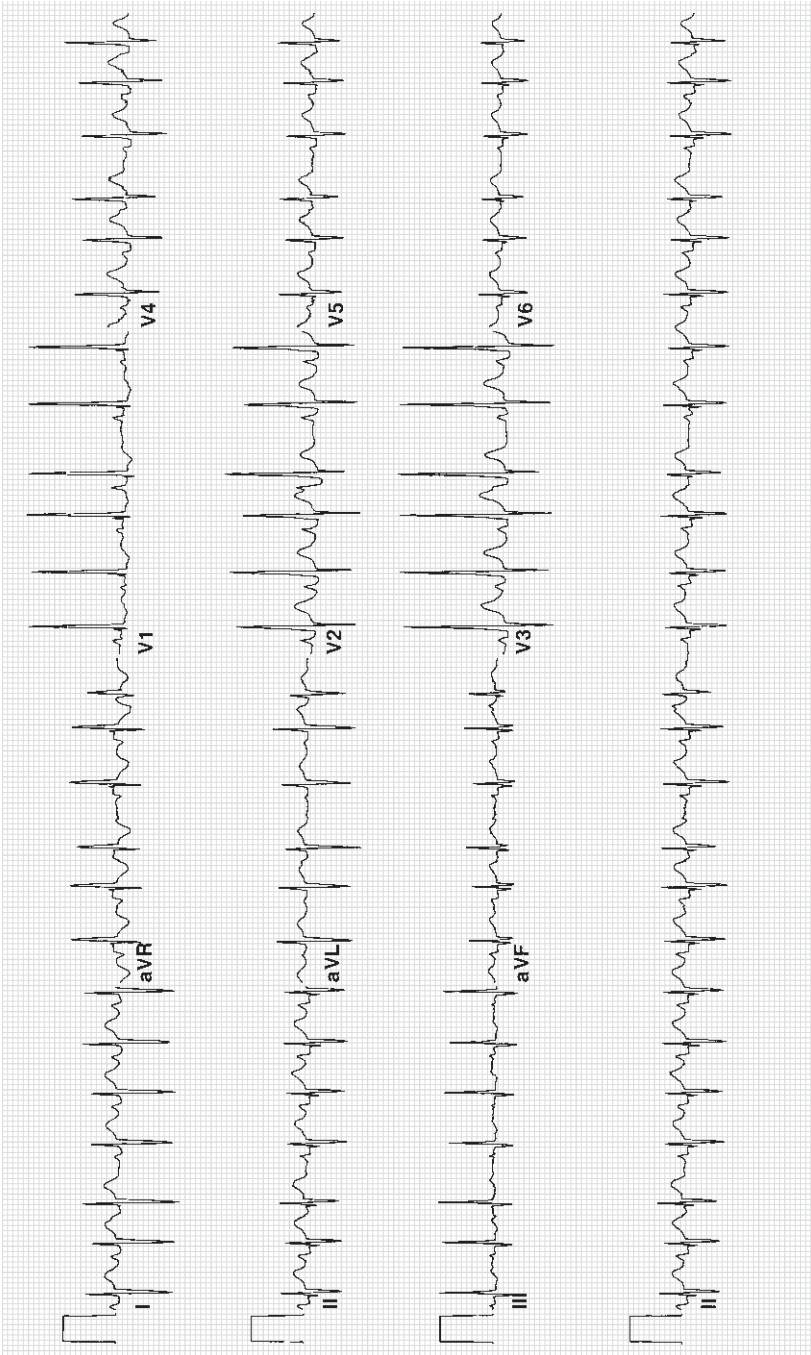


Figure 71 2-week-old infant with cyanosis and irregular heart rhythm.

Figure 71

- Sinus rhythm, 155 bpm
- Frequent premature atrial complexes
- Possible right atrial enlargement
- Right ventricular hypertrophy: rsR' in V1 with R' >15 mm

Premature atrial complexes (PACs)

- Premature P waves differing in axis and morphology from normal sinus P waves
- P wave may be “fused” with preceding T wave
- PR interval may be prolonged with PAC
- Atrioventricular conduction may be normal, aberrant (bundle branch block), or blocked
- Incidence:
 - 50% newborns
 - 14–64% infants, children
- Rarely require treatment

Reference

Kugler, JD. Benign arrhythmias: neonate throughout childhood. In Deal BJ, Wolff GS, Gelband H, eds, *Current Concepts in Diagnosis and Management of Arrhythmia in Infants and Children*. Armonk, NY: Futura Publishing, 1998.

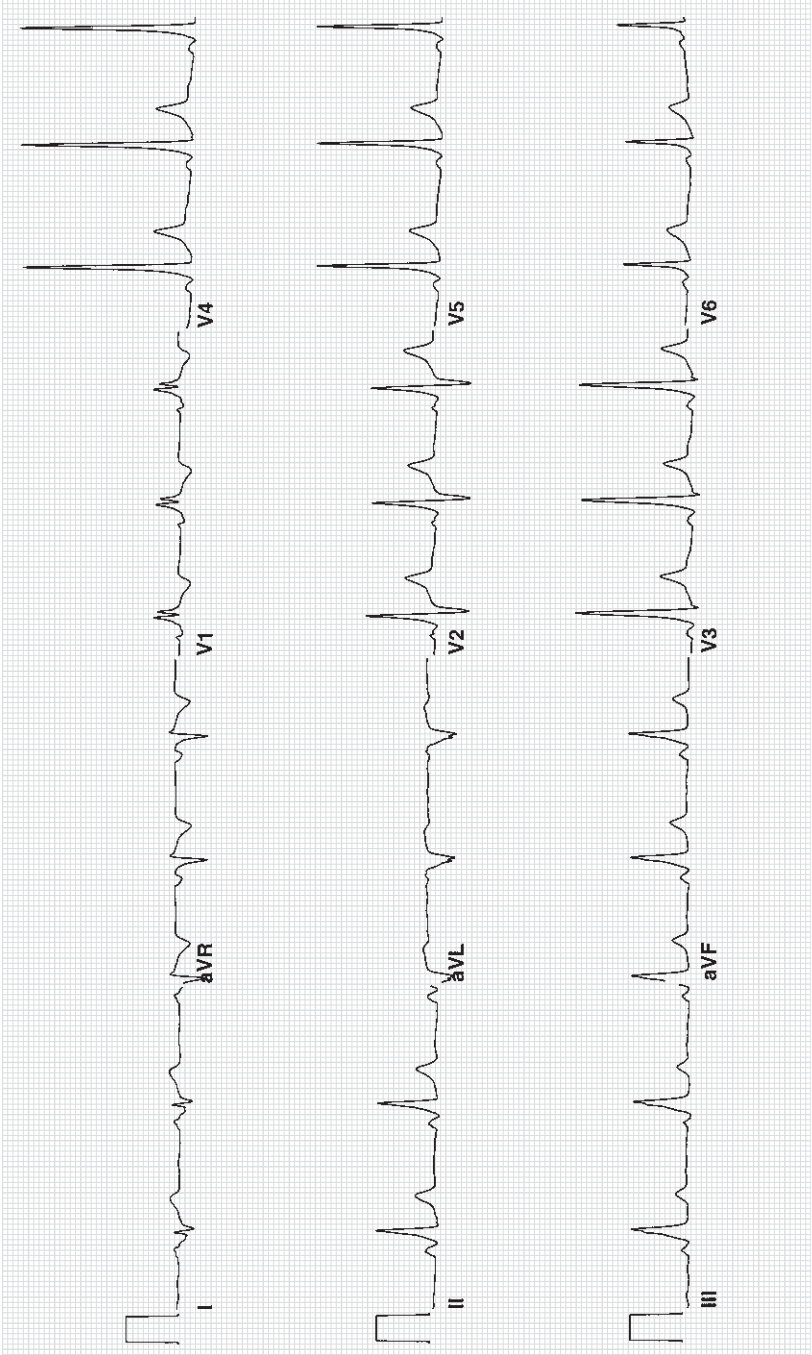


Figure 72 22-year-old male referred for evaluation of palpitations.

Figure 72

- Sinus rhythm, 65 bpm
- Short PR interval = 0.10 msec
- Delta wave with QRS prolongation = 0.15 sec
- Delta wave polarity suggests left lateral accessory connection location
 - (-) in I, aVL
 - (+) in II, III, aVF, V1-V6
 - R/S ratio in V1 > 1

Pre-excitation (Wolff-Parkinson-White pattern)

- PR interval < 0.12 sec with normal P wave
- Prolonged QRS duration for age
- Initial slurred upstroke of QRS complex: delta wave
- Secondary ST and T wave changes
- Accessory atrioventricular connection associated with SVT

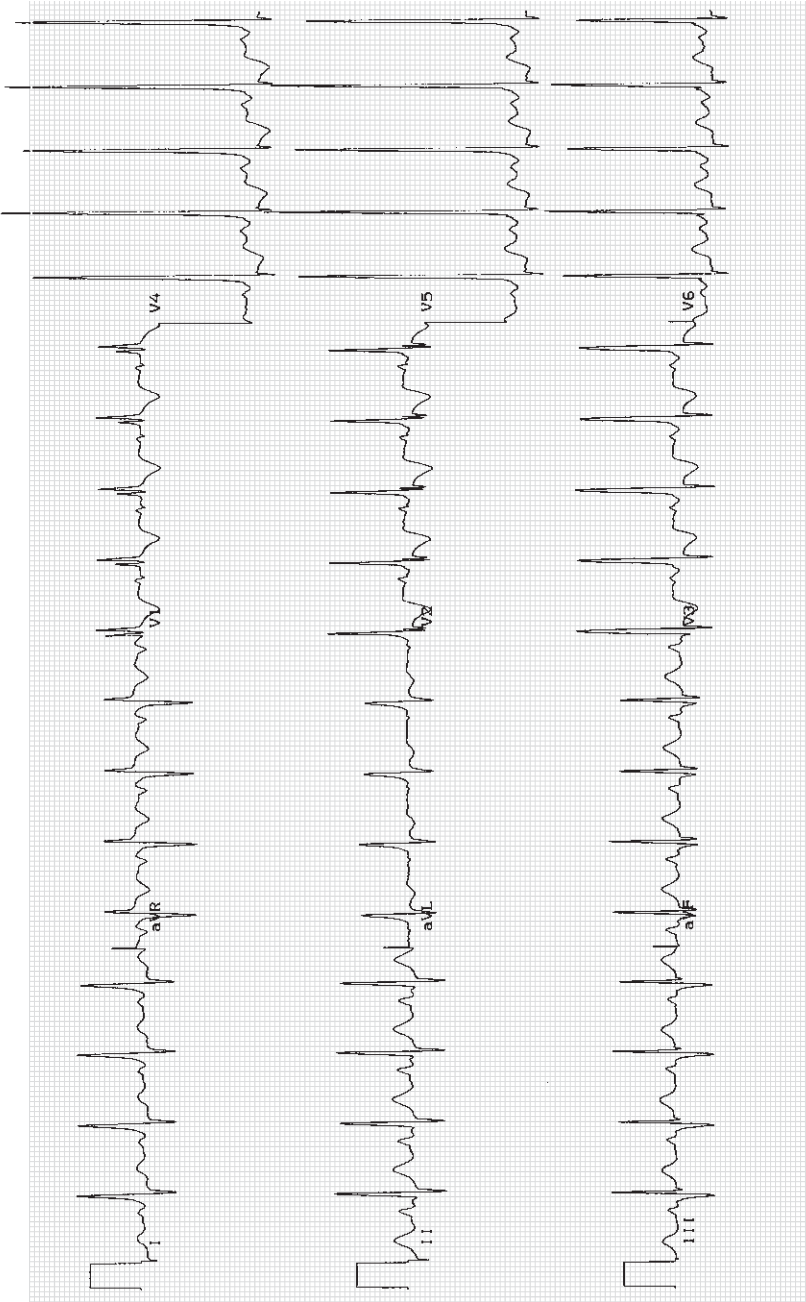


Figure 73 11-year-old boy with palpitations.

Figure 73

- Sinus rhythm, 108 bpm
- Short PR interval = 0.10 sec
- QRS prolongation = 0.12 sec
- Delta wave polarity suggests left posterior accessory connection:
 - (+) in I, aVL, V2–V6
 - (–) in III, aVF
 - R/S ratio in V1 > 1

Wolff–Parkinson–White syndrome

- WPW syndrome:
 - Pre-excitation pattern on ECG plus supraventricular tachycardia
- Incidence 0.1–0.3% of population
- Associated with structural heart disease in up to 20–32% of patients
- 20–30% of patients with pre-excitation on resting ECG will experience SVT

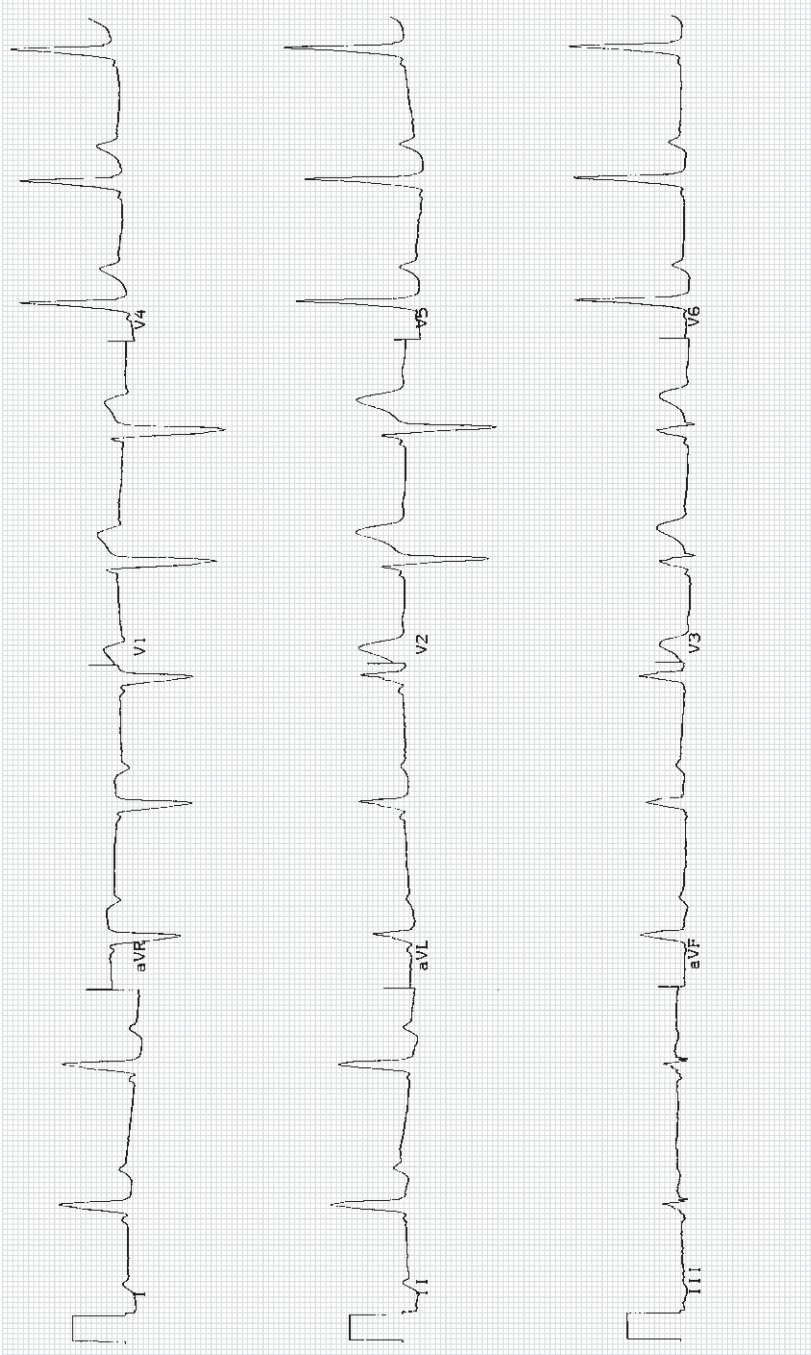


Figure 74 9-year-old boy with palpitations.

Figure 74

- Sinus rhythm, 60 bpm
- Short PR interval = 0.06 sec
- Delta wave with QRS prolongation = 0.15 sec
- Delta wave polarity suggests right anteroseptal accessory connection location:
 - (+) in I, II, aVF
 - R/S ratio in V1 < 1
 - (+/-) in V1
 - R > S in III

Wolff-Parkinson-White Syndrome

- Mechanisms of tachycardia:
 - Orthodromic* reciprocating tachycardia
 - Antidromic** reciprocating tachycardia
 - Atrial tachycardia with antegrade conduction*** over accessory connection

*See p. 169.

**See p. 171.

***See p. 173.

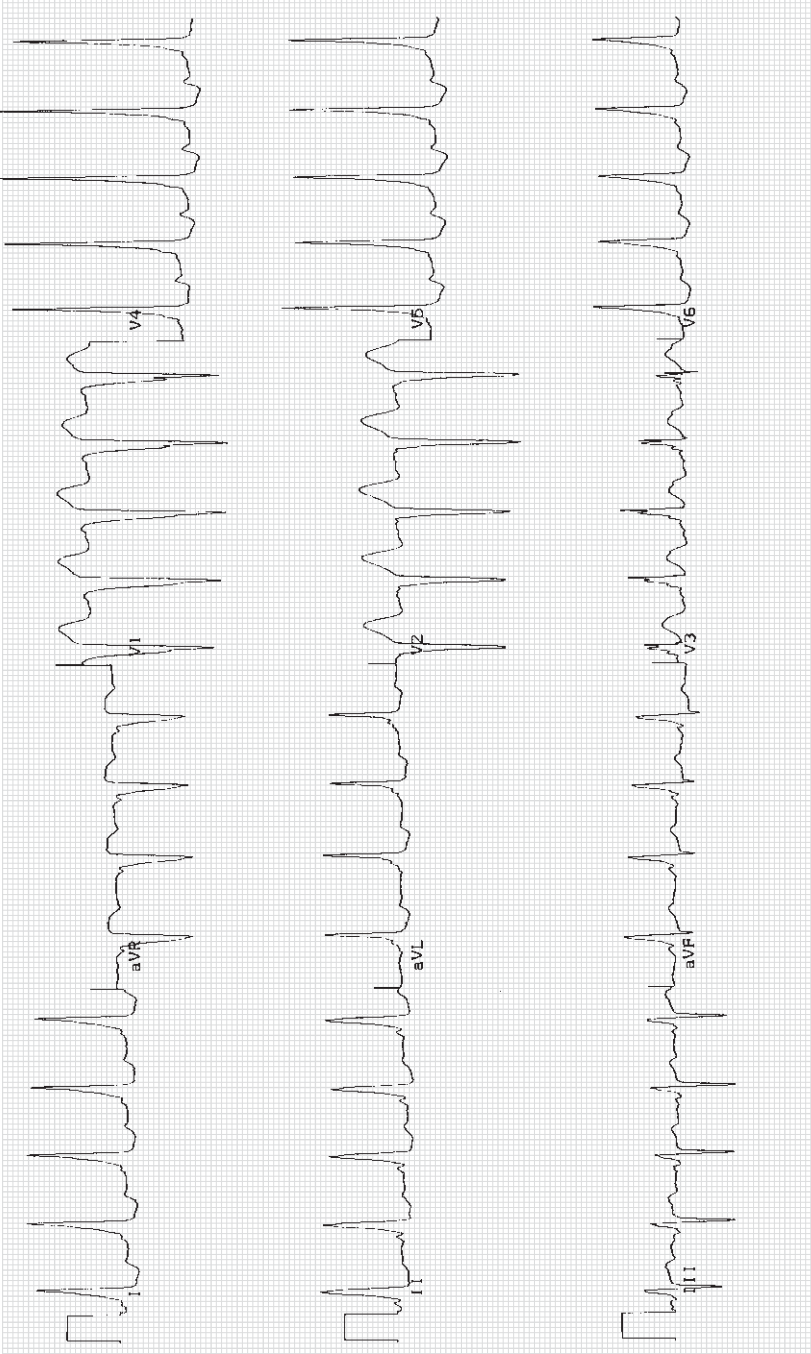


Figure 75 6-year-old boy with frequent episodes of supraventricular tachycardia.

Figure 75

- Sinus rhythm, 110 bpm
- Short PR interval = 0.04 sec
- Delta wave with QRS prolongation = 0.12 sec
- Delta wave polarity suggests right midseptal accessory connection:
 - (+) in I, II, aVL, aVF, V3–V6
 - R/S ratio in V1 < 1
 - (+/-) in lead V1
 - R < S in lead III

Wolff–Parkinson–White Syndrome

- Right midseptal accessory connection location may be associated with increased risk of heart block during catheter ablation procedures

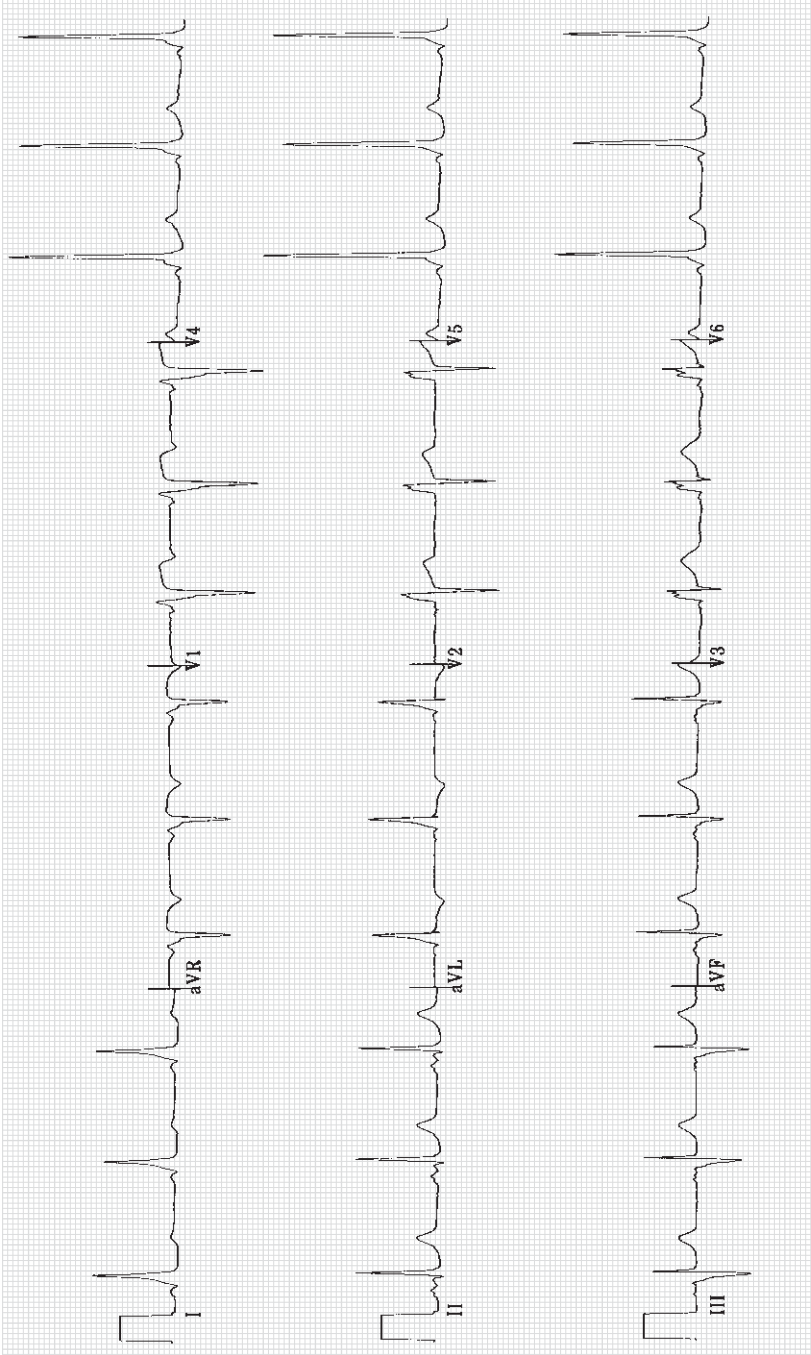


Figure 76 12-year-old boy with syncope during a baseball game.

Figure 76

- Sinus rhythm, 70 bpm
- Short PR interval = 0.06 sec
- Delta wave with QRS prolongation = 0.16 sec
- Delta wave polarity suggests right posteroseptal accessory connection location:
 - (+) in I, aVL, V2–V6
 - R/S ratio in V1 < 1
 - (+/-) in V1
 - (-) in III, aVF

Wolff–Parkinson–White syndrome

- Wide/deep Q wave reflects pre-excitation, not myocardial infarction
- Right-sided accessory connections:
 - May be associated with Ebstein's anomaly of the tricuspid valve and may have multiple accessory connections

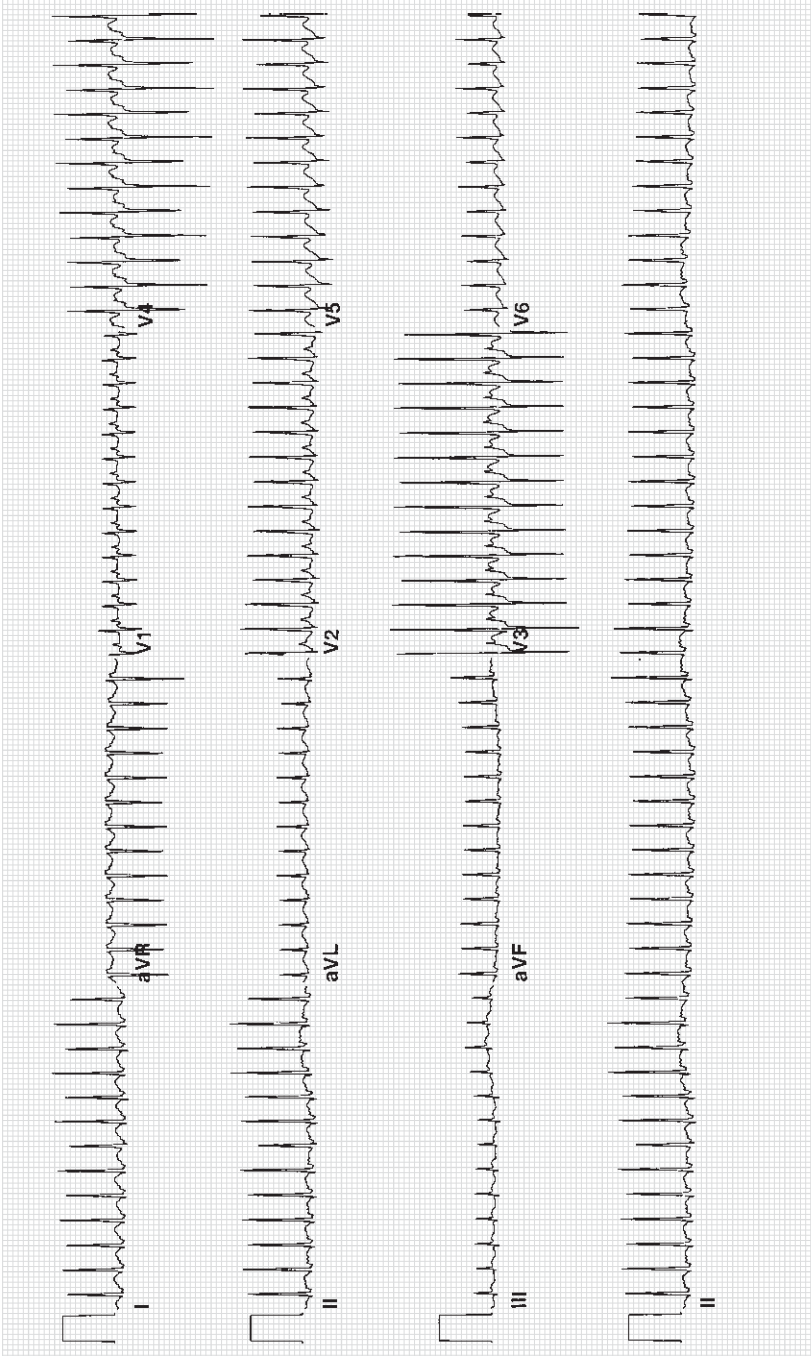


Figure 77 6-day-old infant with tachycardia and irritability.

Figure 77

- Narrow regular QRS tachycardia, 320 bpm
- Retrograde P wave follows QRS:
Best seen in leads II, III, aVF, V1, V2
QRS–P interval = 0.08 sec
- Nonspecific ST segment and T wave changes

Orthodromic reciprocating tachycardia

- Supraventricular tachycardia usually rates 150–300 bpm (neonates, mean rate 280 bpm)
- Usually regular, narrow QRS morphology, although rate-related bundle branch block occurs in 15–30% of episodes (often apparent at onset of SVT)
- Retrograde P waves following QRS, with RP interval ≥ 0.07 sec
- Terminates in presence of atrioventricular block
- Reentrant rhythm: antegrade conduction over atrioventricular node to ventricle, retrograde conduction to atria via accessory connection
- Accounts for up to 80% of SVT in infants and young children

Reference

Deal BJ. Supraventricular tachycardia mechanisms and natural history. In *Current Concepts in Diagnosis and Management of Arrhythmias in Infants and Children*. Armonk, NY: Futura Publishing, 1998, 119–126.

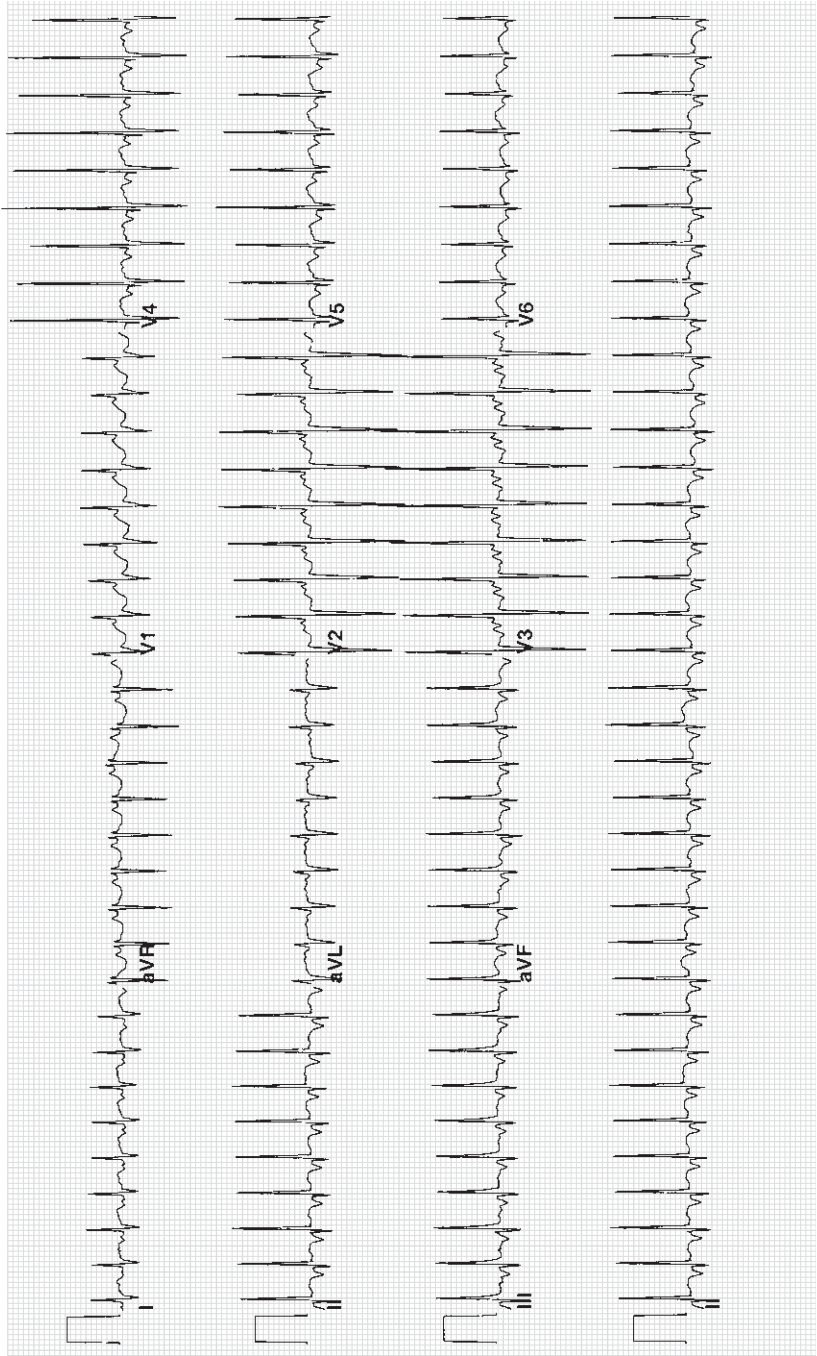


Figure 78 1-month-old infant with poor feeding and tachypnea.

Figure 78

- Regular, narrow QRS tachycardia, 215bpm
- Negative P wave in leads II, III aVF, V6:
P wave precedes QRS
Long R-P interval = 0.18 sec
RP interval > PR interval

Permanent form of junctional reciprocating tachycardia (PJRT)

- Usually regular, narrow QRS tachycardia, rates 130–220 bpm
- Catecholamine sensitivity results in rate variability with exertion or stress
- Retrograde P waves with long RP interval, usually >0.15 sec
- Typically inverted P waves in II, III, aVF
- A variant of orthodromic reciprocating tachycardia, with slowly conducting retrograde accessory connection
- Often incessant, may be associated with heart failure or cardiomyopathy
- Differential diagnosis:
 - Automatic (ectopic) atrial tachycardia
 - Atypical atrioventricular nodal re-entry tachycardia

Reference

Deal BJ. Supraventricular tachycardia mechanisms and natural history. In *Current Concepts in Diagnosis and Management of Arrhythmias in Infants and Children*. Armonk, NY: Futura Publishing, 1998, 120–122.

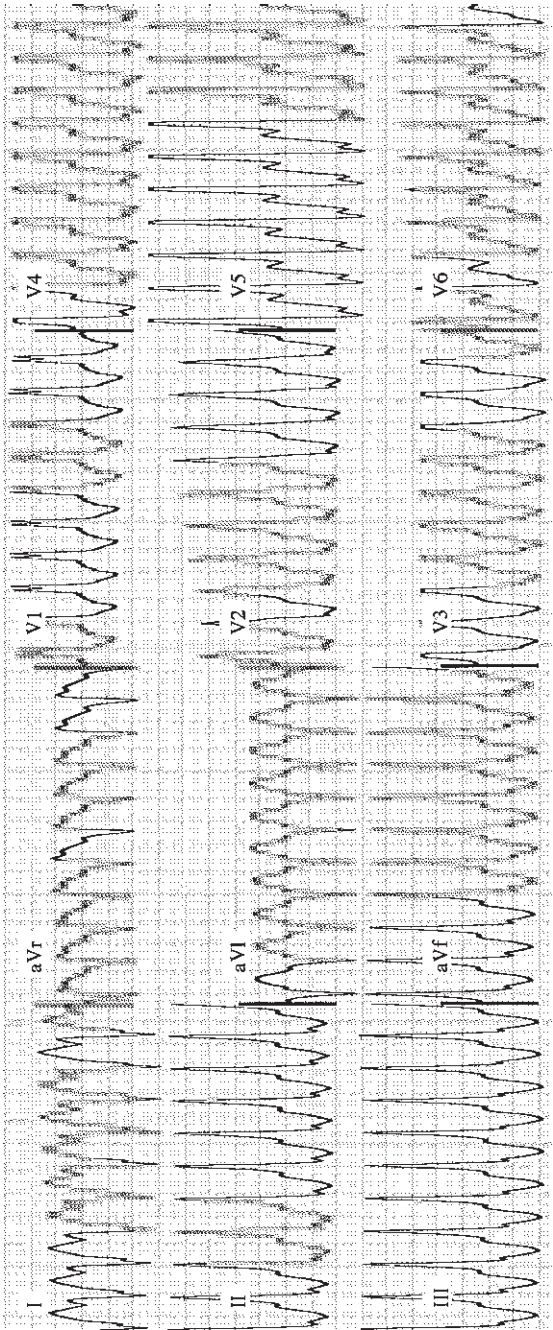


Figure 79 3 year old with tachycardia and history of Wolff–Parkinson–White syndrome, receiving digoxin.

Figure 79

- Regular, wide QRS tachycardia, 240 bpm:
Upright P waves precede QRS, best seen in lead II
PR interval = 0.10 sec
- Right axis deviation, QRS axis $+110^\circ$
- Right bundle branch block pattern:
QRS duration = 0.11 sec
Slurred upstroke of R wave
Antidromic conduction

Antidromic reciprocating tachycardia

- Regular, wide QRS tachycardia
- QRS morphology resembles pre-excited QRS morphology during sinus rhythm (see next tracing on p. 175)
- 1 : 1 AV relationship; tachycardia terminates in presence of AV block
- Re-entrant rhythm: antegrade conduction to ventricle via accessory connection, retrograde conduction to atria via AV node.
- Accounts for less than 10% of SVT associated with accessory connections (Wolff–Parkinson–White syndrome)

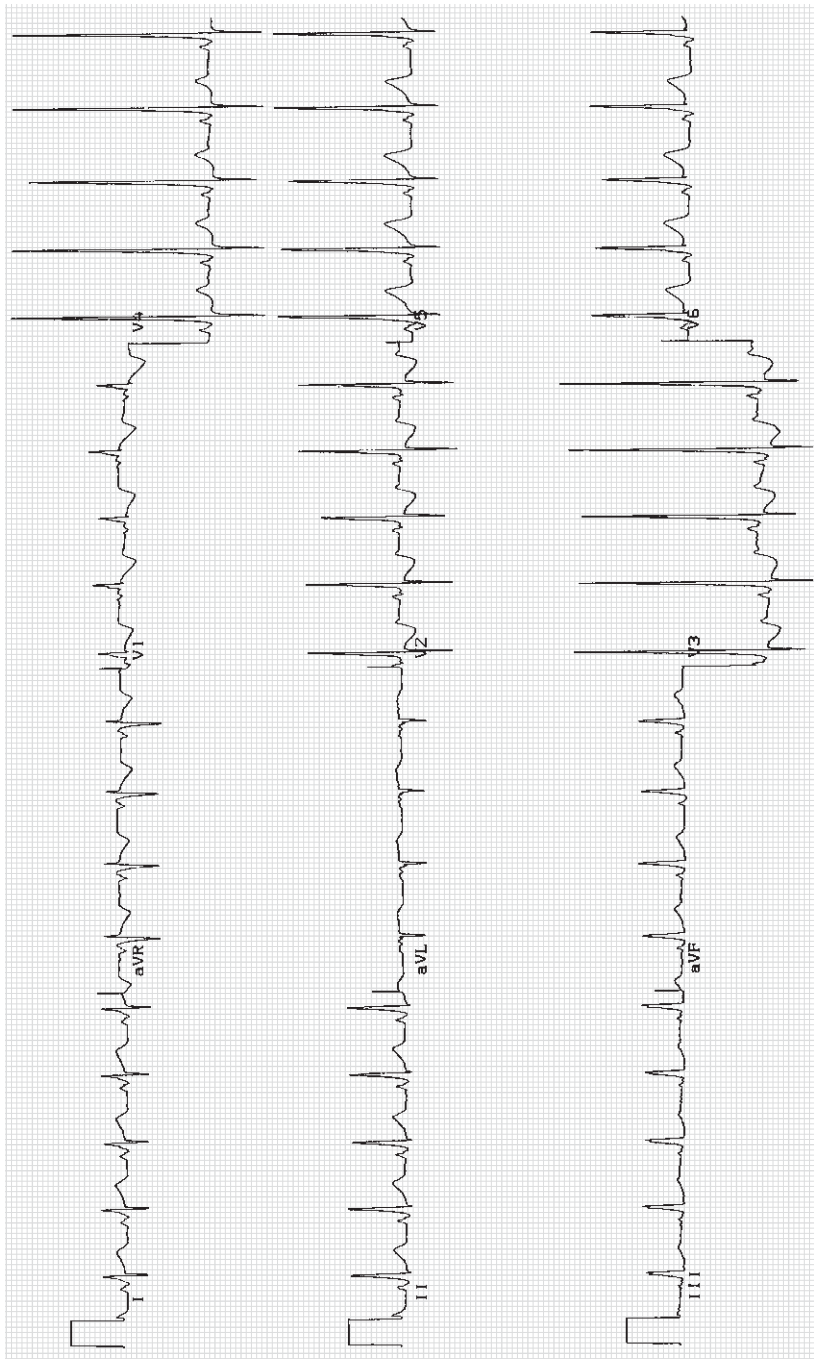


Figure 80 ECG during sinus rhythm of patient (at 2 weeks of age) seen on preceding tracing.

Figure 80

- Sinus rhythm, 113 bpm
- QRS axis $+80^\circ$
- Minimal pre-excitation pattern:
PR interval = 0.08 sec
QRS duration = 0.80 sec
Delta wave polarity:
 (+) in leads I, II, aVF, V4–V6
 (–) in aVL

Pre-excitation

- Compare QRS morphology during sinus rhythm with that seen during antidromic reciprocating tachycardia on prior tracing
- Mild degree of pre-excitation during sinus rhythm, versus maximal pre-excitation during antidromic tachycardia

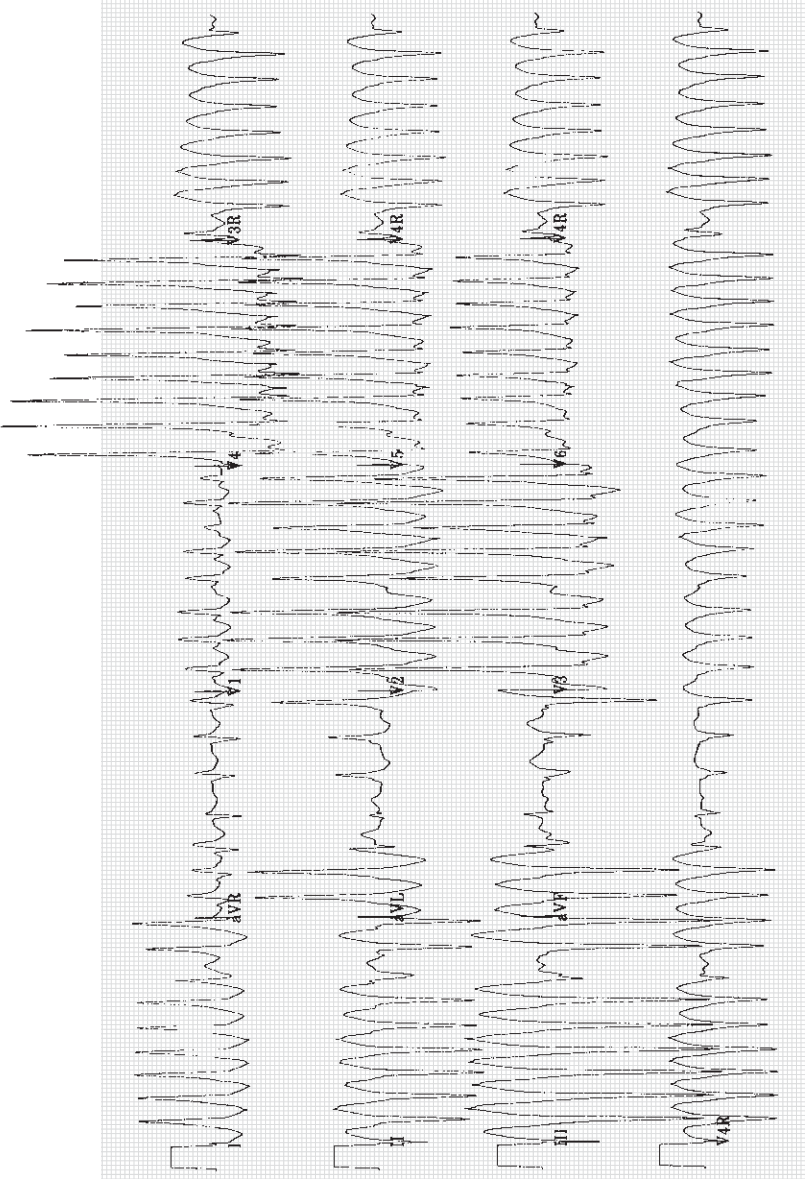


Figure 81 13-year-old male with lightheadedness during exertion. (ECG courtesy of J. Patel, M.D.)

Figure 81

- Irregular, wide QRS tachycardia, 165–247 bpm:
P waves not clearly seen
Variable ventricular rate
Minimal pre-excited RR interval = 0.20 sec
- Left axis deviation
- Prolonged QRS duration:
Tall R wave in both V1 and V6
- Variable QRS morphology

Pre-excited atrial fibrillation

- Atrial fibrillation conducts to ventricles via accessory connection (pre-excited)
- QRS morphology resembles pre-excited QRS during sinus rhythm
- Rapid antegrade conduction via accessory connection to ventricles may precipitate ventricular fibrillation

Reference

Klein GJ, Bashore TM, Sellers TD, Pritchett EL, Smith WM, Gallagher JJ. Ventricular fibrillation in the Wolff-Parkinson-White syndrome. *N Engl J Med* 1979;**310**(20):1080–1085.

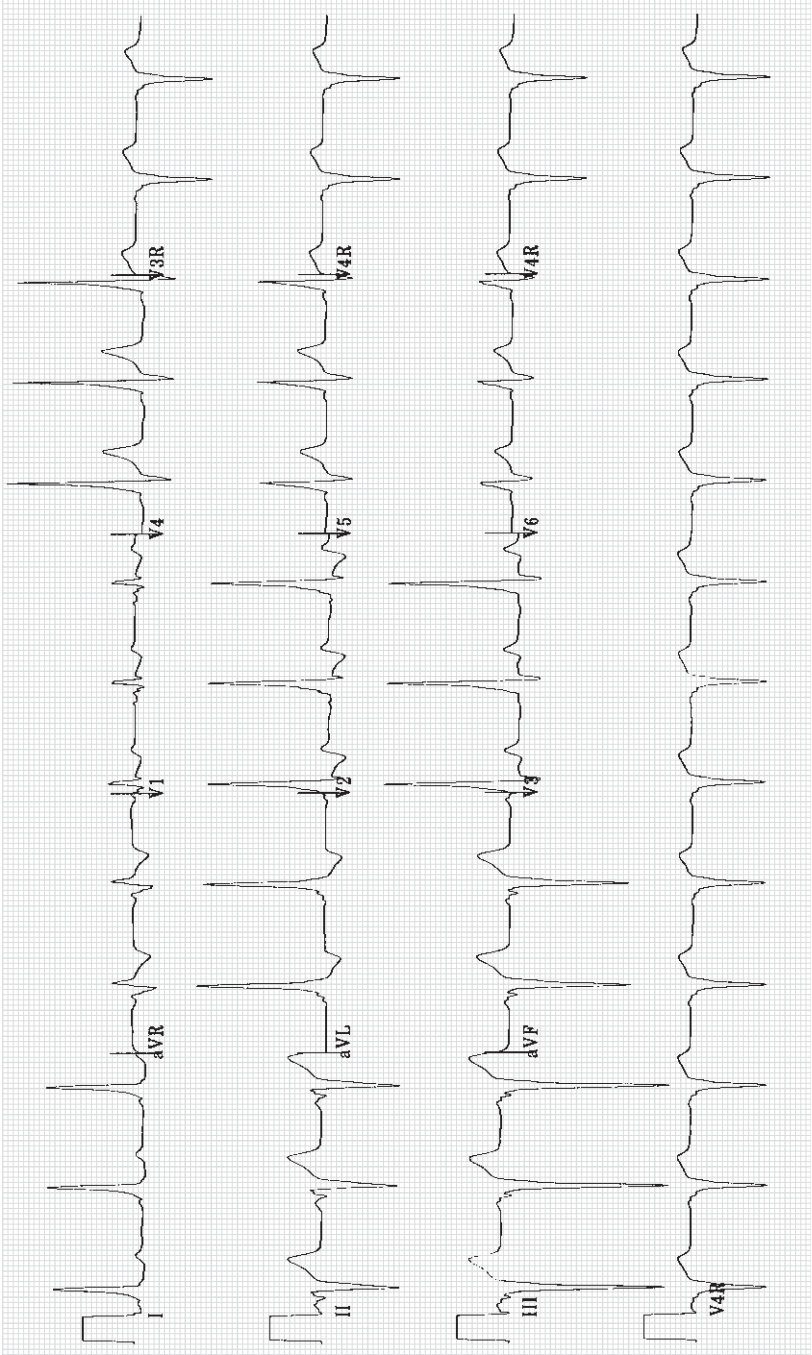


Figure 82 ECG during sinus rhythm of patient seen on preceding tracing. (ECG courtesy of J. Patel, M.D.)

Figure 82

- Sinus rhythm, 77 bpm
- Left axis deviation
- Pre-excitation:

PR interval = 0.08 sec

QRS duration = 0.16 sec

Delta wave polarity:

(+) in I, aVL, V2–V6

(–) in III, aVF, V3R, V4R

Suggests left posterior accessory connection location

Pre-excitation

- Compare QRS morphology during sinus rhythm with that seen during atrial fibrillation on prior tracing
- Varying degrees of pre-excitation during atrial fibrillation accounts for variable QRS morphology

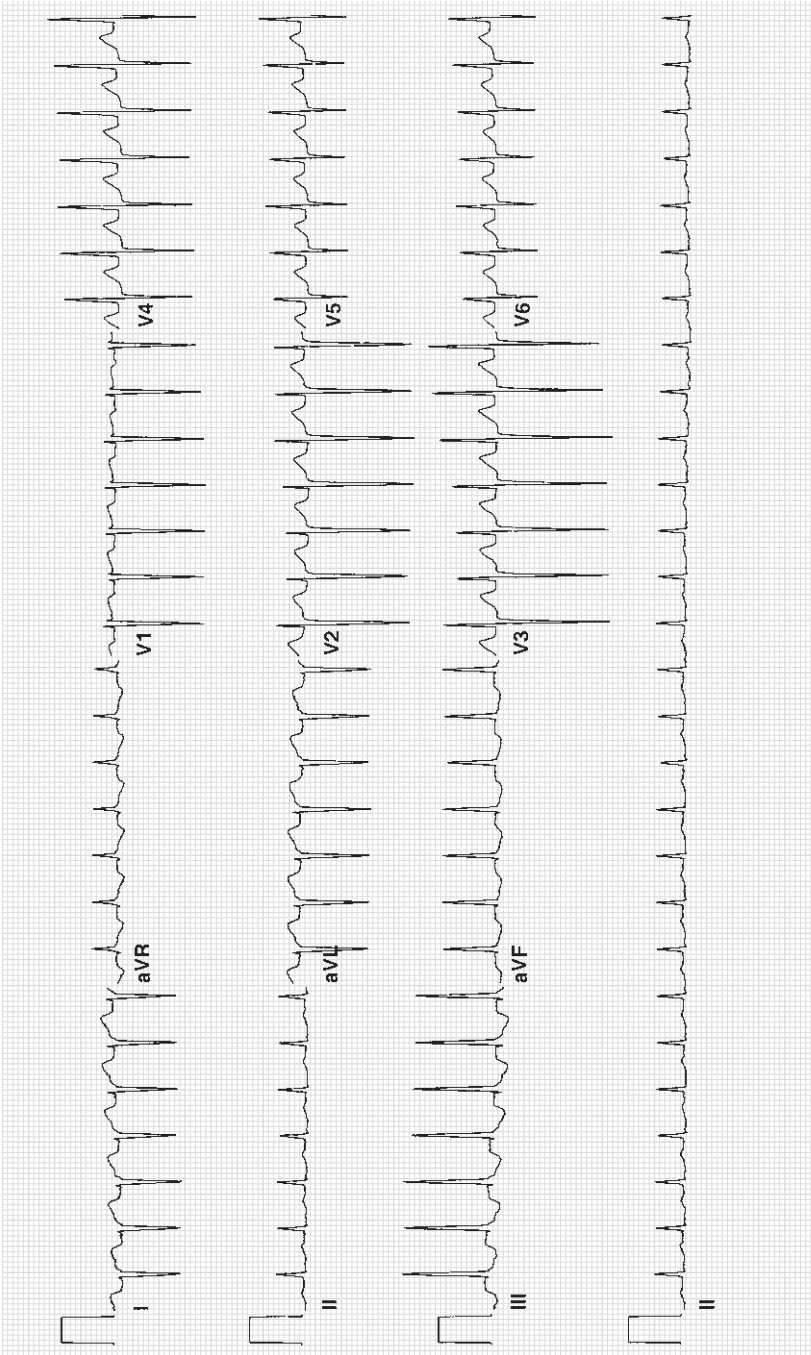


Figure 83 8-year-old girl with brief episodes of tachycardia, terminated with Valsalva maneuver.

Figure 83

- Narrow QRS tachycardia, 170 bpm
- Retrograde P waves difficult to visualize: Best seen in leads III and V1 at end of QRS rsR' pattern in V1
- Right axis deviation
- Possible right ventricular hypertrophy: S wave in V6 = 7 mm

Atrioventricular nodal re-entry tachycardia: Typical ("slow-fast") form

- Supraventricular tachycardia rates 150–300 bpm (mean 170 bpm in older patients)
- Paroxysmal regular, narrow QRS tachycardia
- Retrograde P waves within QRS or at terminal end of QRS:
 - RP interval <0.07 sec
 - Retrograde P wave simulates an S wave in leads II, III, aVF, and r' in V1
- SVT may persist with 2:1 AV conduction, although uncommon
- Reentrant rhythm: antegrade conduction via "slow pathway" of AV node, retrograde conduction via "fast pathway" of AV node
- Rarely seen in infants; by age 6–10 years, accounts for ~30% of SVT, and ~50% of SVT in adults

Reference

Deal BJ. Supraventricular tachycardia: mechanisms and natural history. In *Current Concepts in Diagnosis and Management of Arrhythmias in Infants and Children*. Armonk, NY: Futura Publishing, 1998, 126–128.

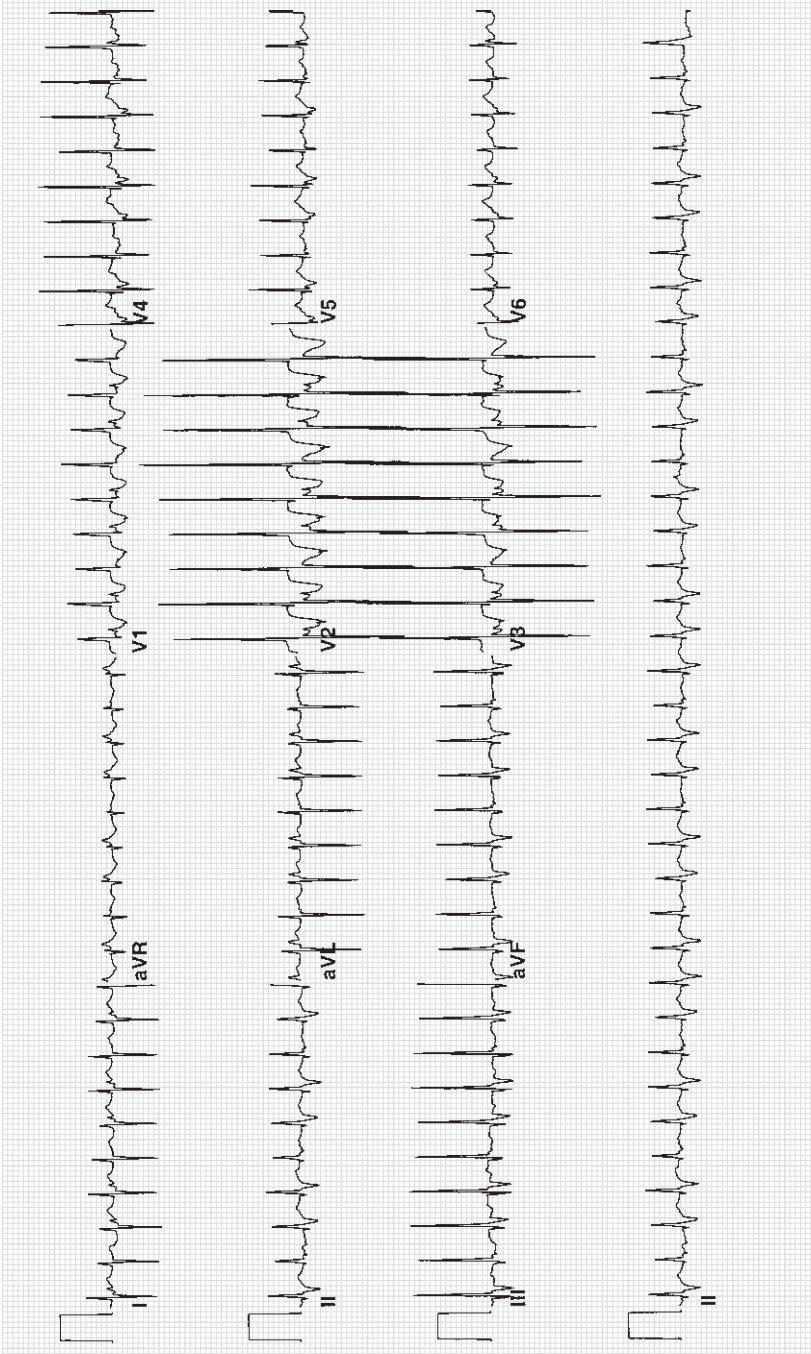


Figure 84 1-month-old infant with incessant tachycardia noted during well-child examination.

Figure 84

- Narrow QRS tachycardia, 226 bpm
- Retrograde P waves best seen in leads aVL, V1–V3, with retrograde Wenckebach conduction

Junctional ectopic tachycardia (JET)

- Tachycardia rates 160–300 bpm, may vary with adrenergic tone
- Usually narrow QRS tachycardia, resembles patient's intrinsic QRS complex
- P waves usually dissociated from QRS, or with retrograde VA conduction
- Due to enhanced automaticity in AV node or His bundle
- Typically seen acutely after surgery near atrioventricular junction (ventricular septal defect, tetralogy of Fallot, Fontan); transient, usually lasting 48–72 hours
- Congenital JET seen rarely in infants, difficult to control, and associated with significant mortality

References

- Walsh EP, Saul JP, Sholler GF, *et al.* Evaluation of a staged treatment protocol for rapid automatic junctional tachycardia after operation for congenital heart disease. *J Am Coll Cardiol* 1997;**29**:1046–1053.
- Villain E, Vetter VL, Garcia JM, Herre J, Cifarelli A, Garson A Jr. Evolving concepts in the management of congenital junctional ectopic tachycardia. A multicenter study. *Circulation* 1990;**81**(5):1713–1714.

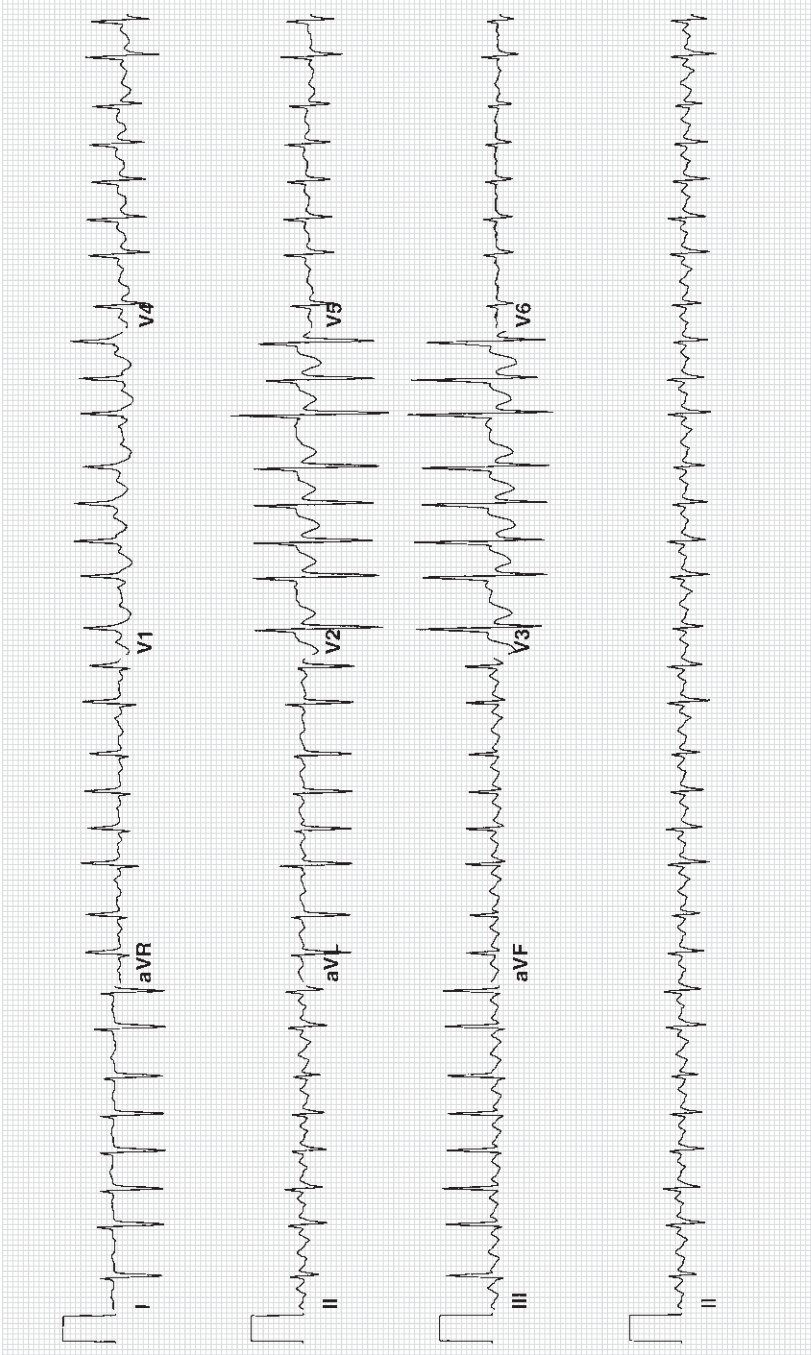


Figure 85 1-day-old infant with tachycardia *in utero*.

Figure 85

- Irregular narrow QRS rhythm, ventricular rate 193 bpm
- “Sawtooth” atrial flutter waves:
 - Best seen in leads II, III, aVF, V1, V2
 - Atrial rate ~400 bpm
- Variable atrioventricular conduction (2:1–3:1)

Atrial flutter

- Atrial rates 250–480 bpm
- Rapid, regular sawtooth flutter waves (monomorphic negative or biphasic P waves in leads II, III, aVF)
- Variable AV conduction, usually 2:1
- Bimodal age distribution in pediatric patients:
 - Neonate (or fetus) with structurally normal heart
 - Older patient with postoperative congenital heart disease (atrial septal defect, Fontan, Mustard or Senning, tetralogy of Fallot, mitral valve disease)
- May be associated with sick sinus syndrome in postoperative patients

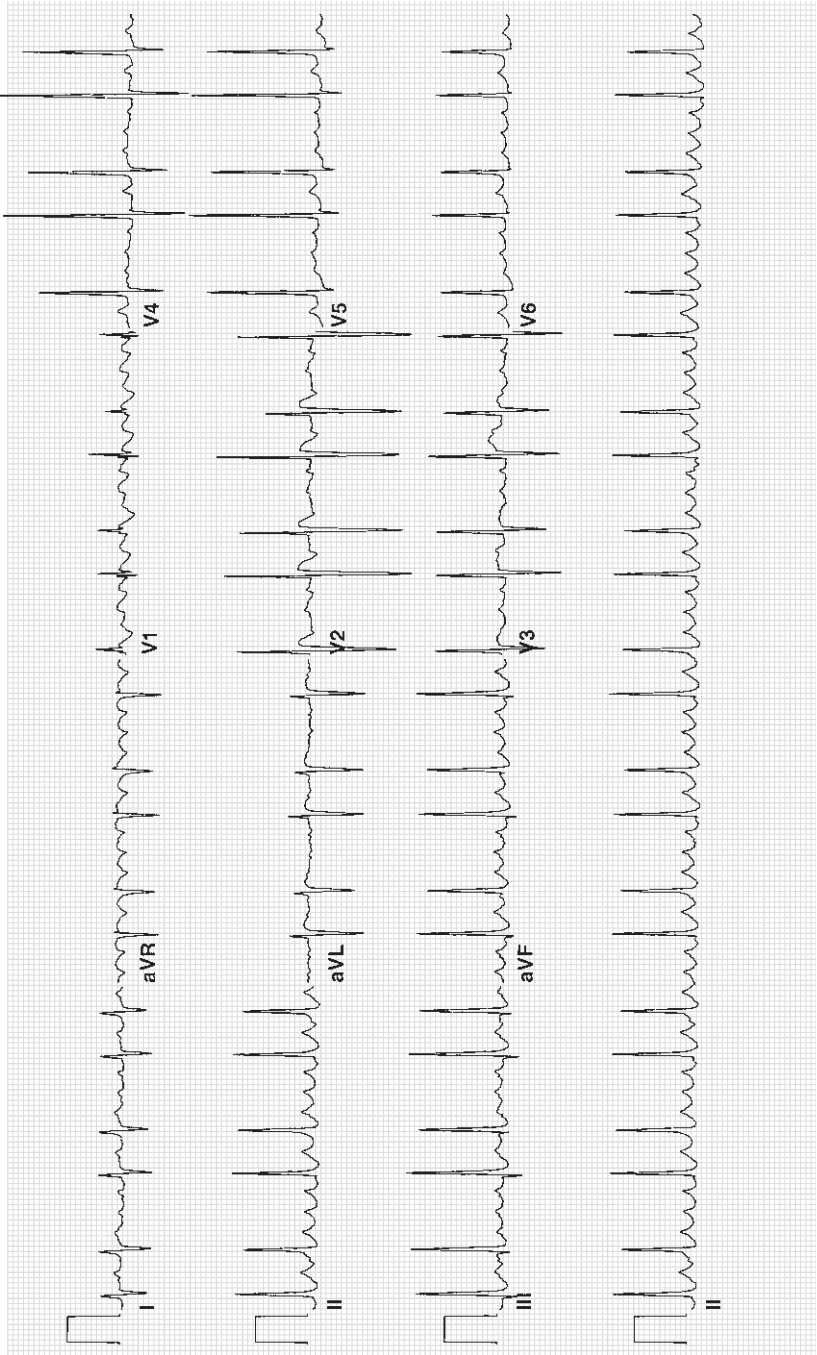


Figure 86 4-week-old infant, status post-arterial switch operation for transposition of the great arteries.

Figure 86

- Irregular narrow QRS tachycardia, ventricular rate averages 130 bpm
- Atrial tachycardia with distinct P waves, best seen in leads I, II, III, aVF, V1
- Variable AV conduction (2:1 and 4:1)

Atrial re-entry tachycardia: Acute postoperative setting

- Distinct P waves, atrial rates 200–300 bpm
- PR interval may be prolonged
- Atrioventricular conduction typically 2:1; variable conduction may produce irregular ventricular response
- Atrial tachycardia persists with atrioventricular block
- Note: Usually slower atrial rates and distinct P waves (absence of flutter waves) distinguishes atrial re-entry from atrial flutter
- Suspect in acute postoperative setting with *fixed* HR ≥ 150 bpm

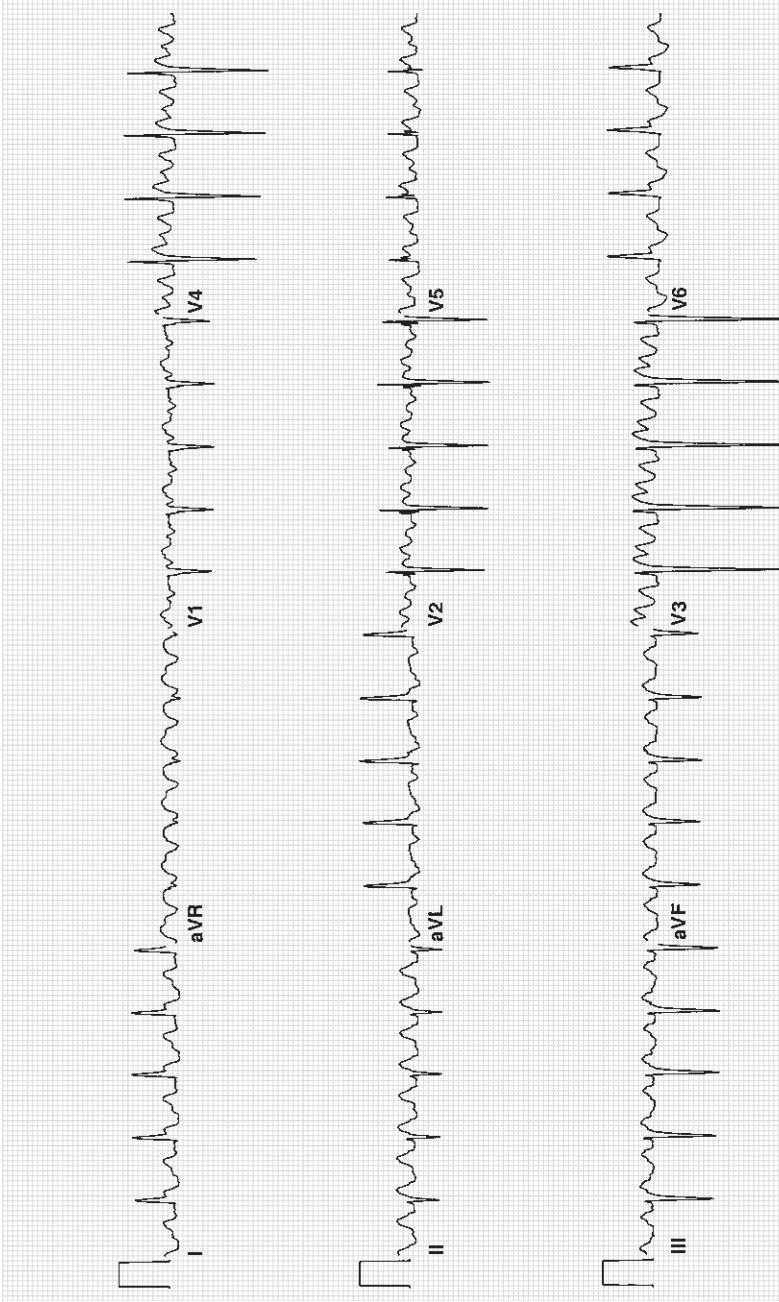


Figure 87 10-year-old girl, status post-Fontan procedure for tricuspid atresia, in emergency room for headache and fatigue.

Figure 87

- Regular narrow QRS tachycardia, ventricular rate 120 bpm
- Distinct P waves seen in leads II, III, aVF; atrial rate 240 bpm
- 2:1 atrioventricular conduction
- Left axis deviation: QRS axis -40°
- Decreased right ventricular voltage

**Atrial re-entry tachycardia:
Late postoperative setting**

- Atrial tachycardia, rates usually <250 bpm
- Similar to atrial flutter, but slower atrial rates, and absence of “sawtooth” flutter waves
- Variable atrioventricular conduction, usually 2:1
- Distinct or low amplitude P wave precedes QRS

Reference

Müller GI, Deal BJ, Strasburger JE, Benson DW. Electrocardiographic features of atrial tachycardias after operation for congenital heart disease. *Am J Cardiol* 1993;71:122–124.

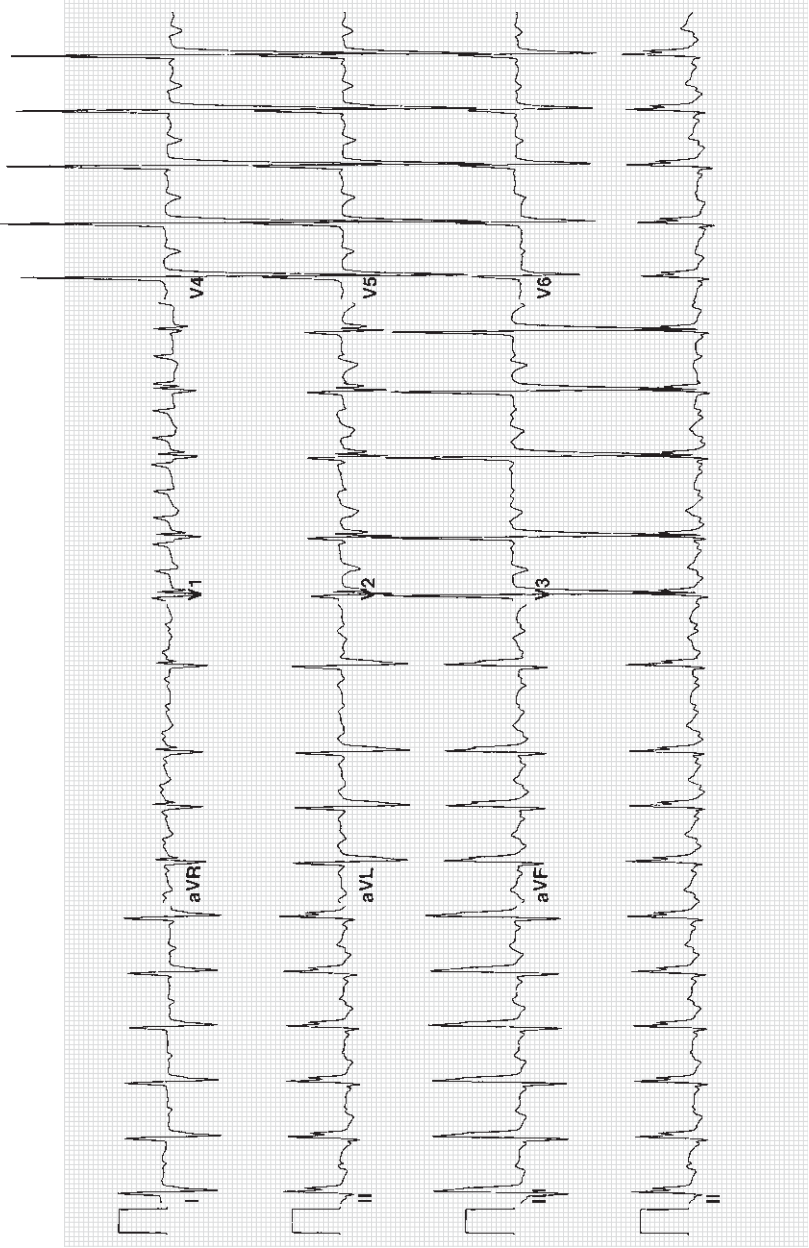


Figure 88 14-year-old boy, status post-Fontan procedure for complex single ventricle; complaining of fatigue.

Figure 88

- Slightly irregular narrow QRS tachycardia, ventricular rate 120 bpm
- Note distinct P waves in lead V1; atrial rate 270 bpm
- Variable atrioventricular conduction, predominantly 2:1
- Ventricular hypertrophy:
 - R + S wave amplitude in V4 > 60 mm
 - S wave in V6 = 16 mm
 - Q wave in V1
- T wave inversion in inferolateral leads

Atrial re-entry tachycardia: Late postoperative setting

- Consider diagnosis whenever HR > 100 bpm in postoperative Fontan, Mustard, Senning patient
- Requires prompt conversion to sinus rhythm
- If untreated, associated with atrial thrombosis, stroke, heart failure, or sudden death
- Suspect in late postoperative setting when HR > 100 bpm

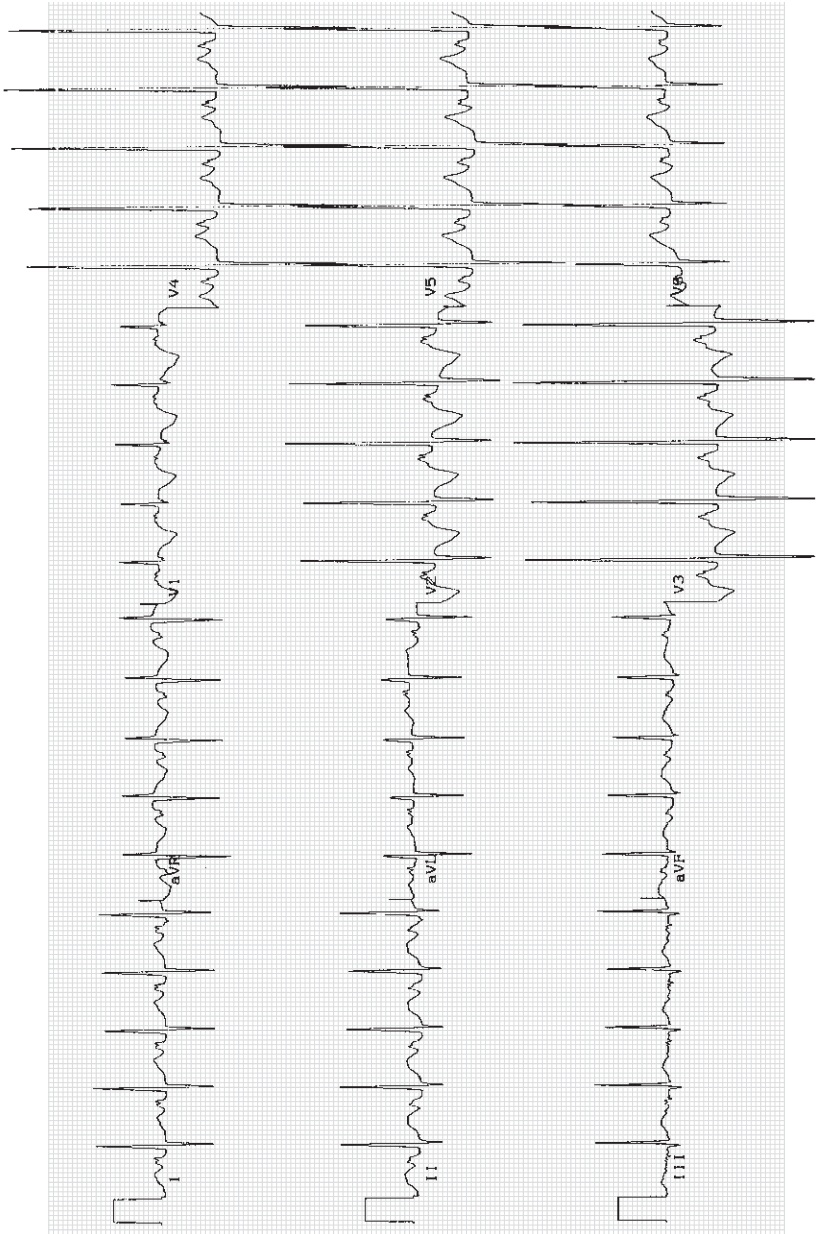


Figure 89 4-month-old infant with congestive heart failure; tachycardia rate slowed by anti-arrhythmic medications.

Figure 89

- Regular narrow QRS tachycardia, 120 bpm
- Notched P wave precedes each QRS
- Combined ventricular hypertrophy:
 - R + S waves in V4 >60 mm
 - S wave in V6 >10 mm
- Prolonged QTc

Automatic atrial tachycardia

- Distinctly visible P wave with abnormal morphology (notching) or abnormal axis compared with sinus P waves
- Prolonged PR interval, especially with faster rates
- Atrial rates 130–280 bpm, with rate variability
- Atrial tachycardia persists with atrioventricular block

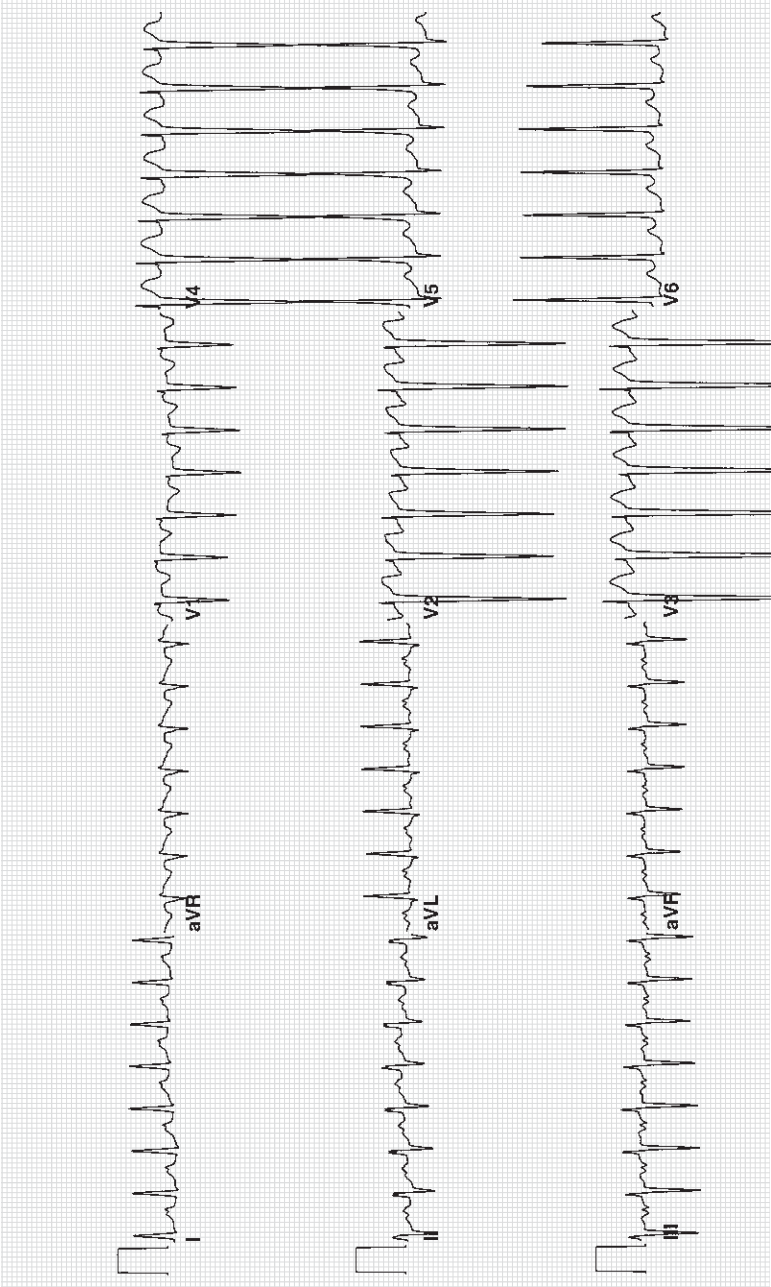


Figure 90 10-year-old girl with nausea, fatigue, and cardiomegaly.

Figure 90

- Regular narrow QRS tachycardia, 175 bpm
- Notched P wave precedes each QRS
- First degree atrioventricular block: PR interval = 0.18 sec
- Left axis deviation QRS axis: -40°
- Left ventricular hypertrophy:
 - R wave in V6 = 28 mm
 - S wave in V2 = 33 mm
 - ST depression in V5, V6

Automatic atrial tachycardia

- Tachycardia is often incessant, may cause dilated cardiomyopathy
- Usually seen in patients with structurally normal heart, less commonly in postoperative patients with congenital heart disease

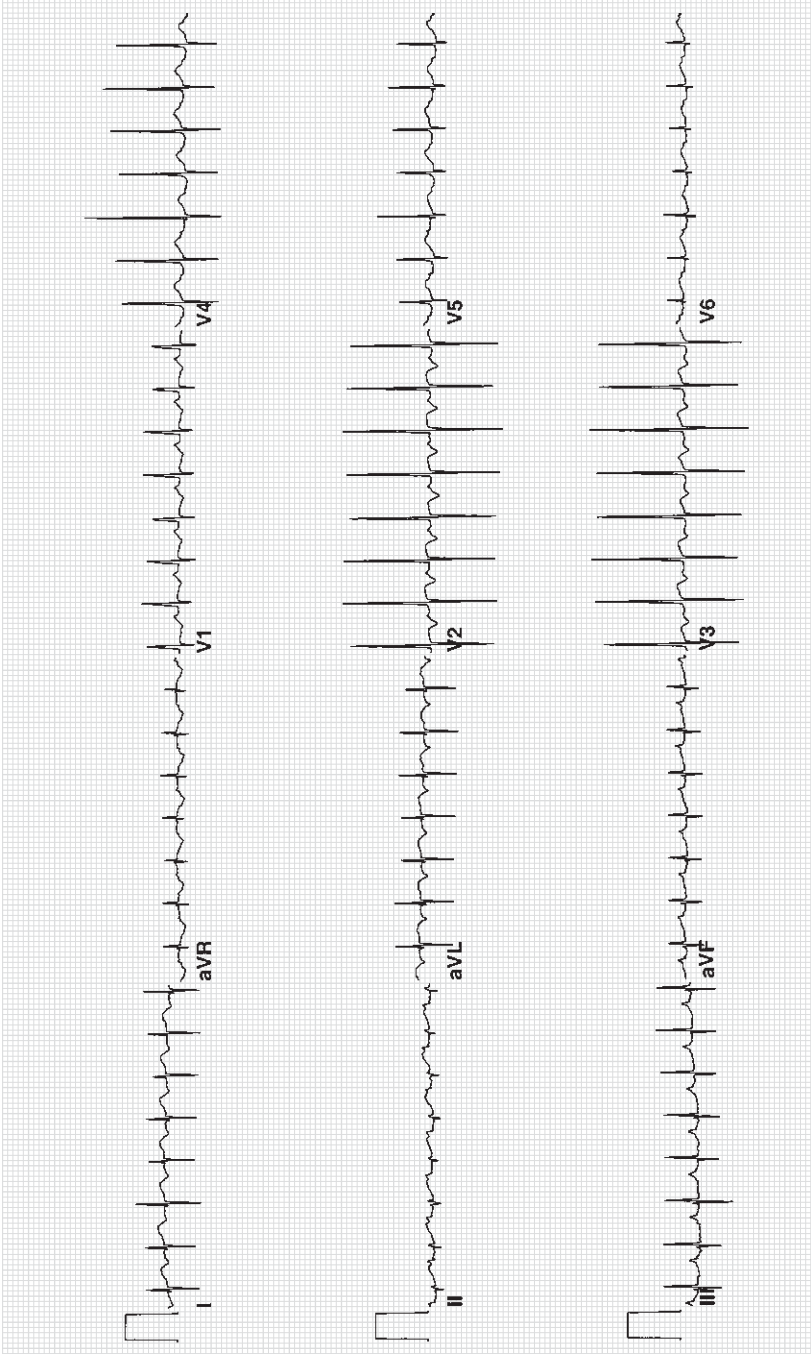


Figure 91 2-week-old infant with tachycardia.

Figure 91

- Regular narrow QRS tachycardia, 182 bpm
- Low amplitude P wave precedes each QRS
- Negative P wave in leads I, aVL suggests left atrial origin

Automatic atrial tachycardia

- Low amplitude P waves in limb leads may be clue to diagnosis
- Consider obtaining ECG in infants with HR >180 bpm

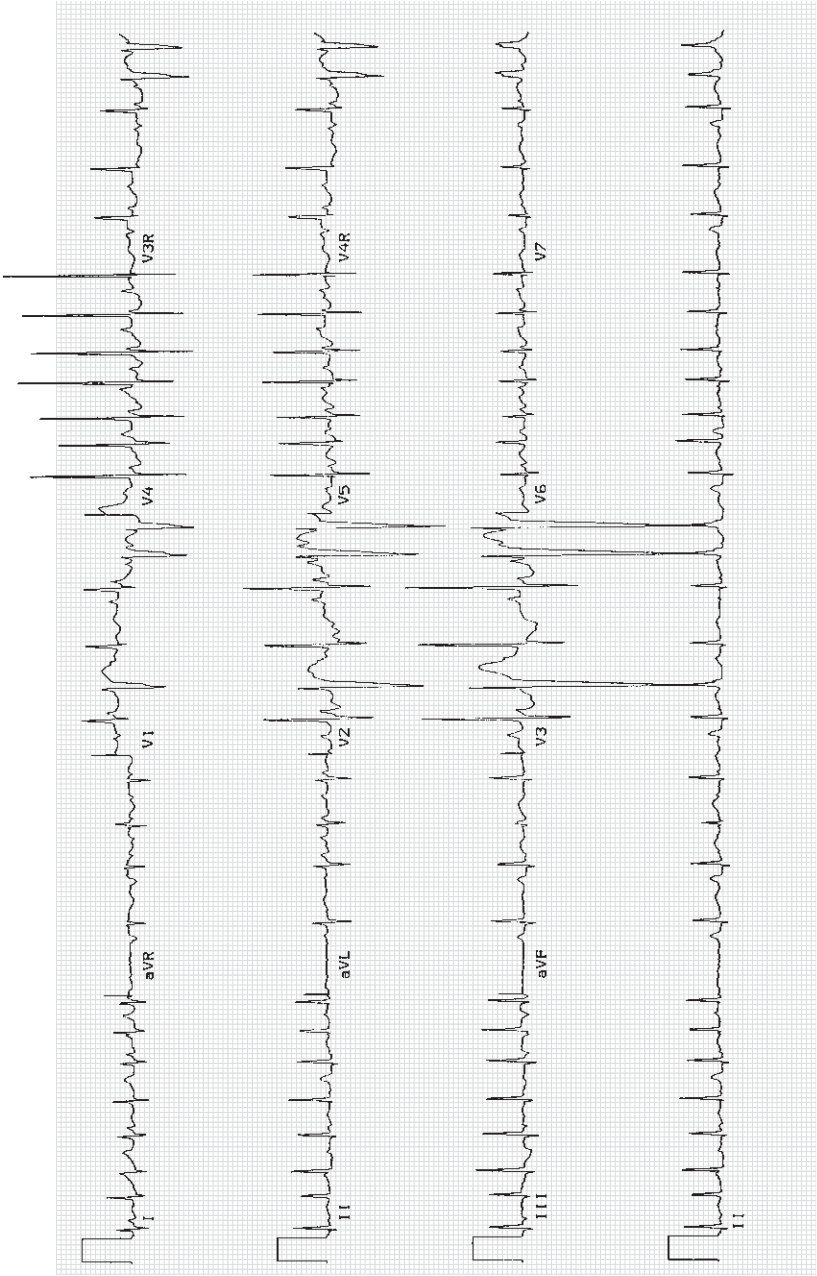


Figure 92 Neonate with irregular tachycardia. (ECG courtesy of Vicki Pyevich, M.D.)

Figure 92

- Irregular tachycardia with narrow and wide QRS complexes, averages 175 bpm
- Multiple distinct P wave morphologies, firing randomly, gives chaotic appearance
- Some P waves conducted aberrantly (long-short coupling, Ashman's phenomenon)

Multifocal atrial tachycardia

- At least three distinct, different, nonsinus P wave morphologies
- Irregular atrial rate >100 bpm
- Isoelectric baseline between P waves
- Varying P-P, PR, and R-R intervals
- Rare arrhythmia, usually seen in infants or adults; in childhood often associated with structural heart disease, dysmorphism, pulmonary or neurologic disease

Reference

Bradley DJ, Fischbach PS, Law IH, Serwer GA, Dick M 2nd. The clinical course of multifocal atrial tachycardia in infants and children. *J Am Coll Cardiol* 2001;**38**:401–8.

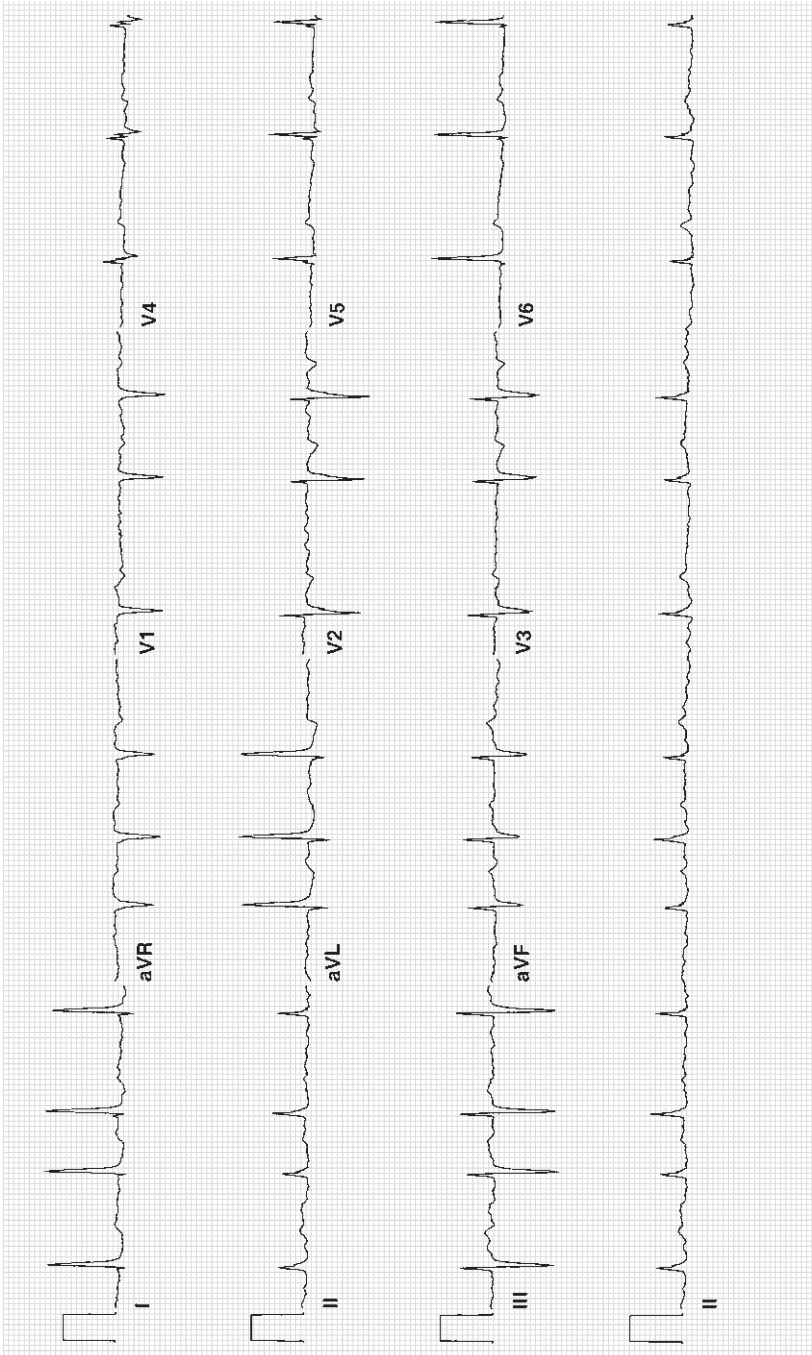


Figure 93 16-year-old girl with palpitations, status post-Fontan procedure.

Figure 93

- Irregular ventricular rate, 75 bpm
- Fine irregular, rapid, chaotic fibrillatory atrial activity best seen in leads II, V1, V2
- Variable atrioventricular conduction
- Left axis deviation: QRS axis -5°
- Decreased R wave amplitude in right precordium
- Nonspecific ST wave abnormality

Atrial fibrillation

- Disorganized atrial activity at rates >350 bpm, with coarse or fine fibrillatory waves best seen in leads V1 and V2
- “Irregularly irregular” ventricular response, due to variable atrioventricular conduction
- Uncommon in childhood; associated with structural heart disease, thyrotoxicosis, SVT due to an accessory connection, high vagal tone, or familial forms

Reference

Deal BJ. Supraventricular tachycardia: mechanisms and natural history. In *Current Concepts in Diagnosis and Management of Arrhythmias in Infants and Children*. Armonk, NY: Futura Publishing, 1998, 133–134.

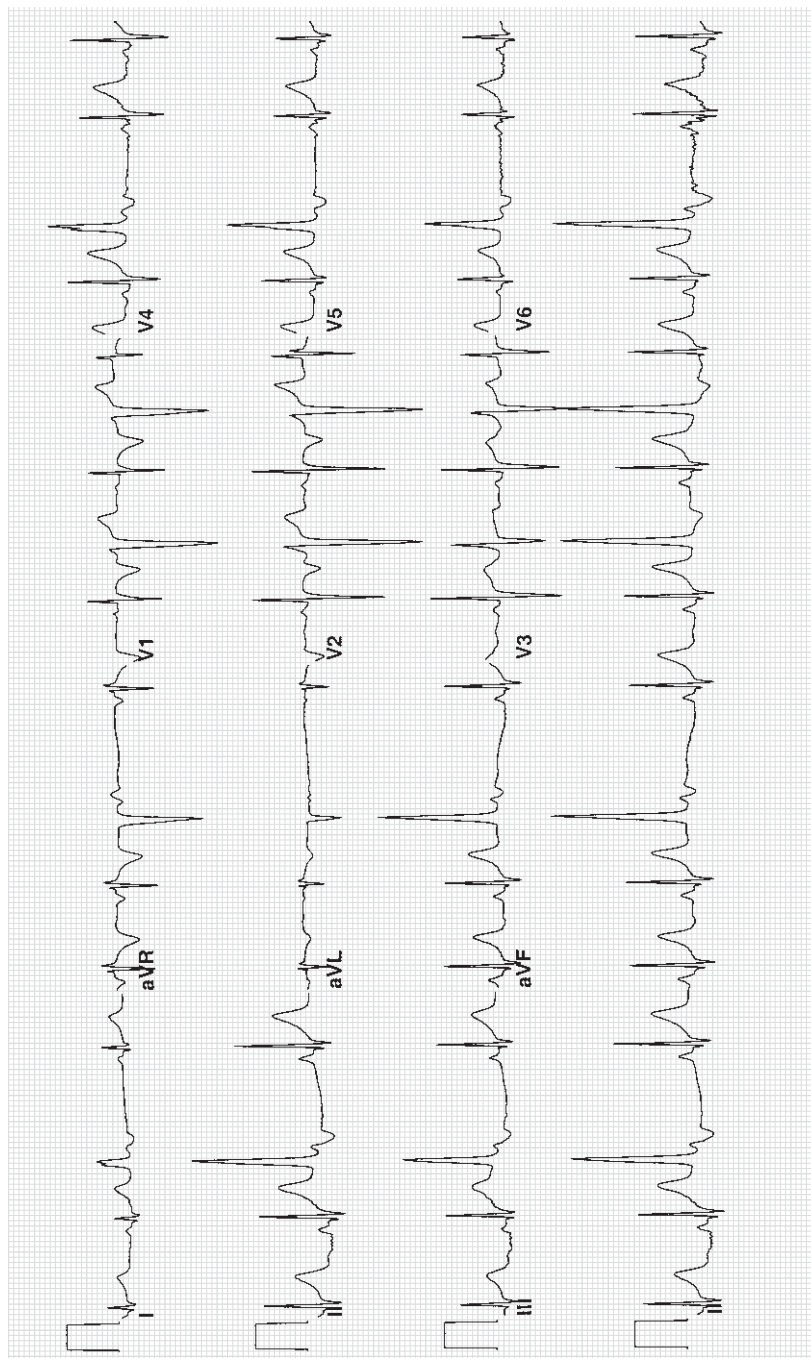


Figure 94 3-year-old boy with irregular heart rhythm.

Figure 94

- Sinus rhythm, 100 bpm
- Frequent premature wide QRS complexes (premature ventricular complexes)
- Note sinus P waves occurring after PVC; “AV dissociation”
PVCs are interpolated into sinus rhythm
- PVC morphology:
Left bundle branch block pattern in V1 suggests origin from right ventricle
Normal axis (negative in aVL) suggests origin from RV outflow tract
- Voltages and intervals of sinus beats are normal

Premature ventricular complexes (PVCs)

- Premature QRS complex without preceding P wave
- QRS morphology differs from sinus conducted beat
- QRS duration usually, but not always, prolonged
- Incidence:
18% newborns
6–8% infants–6 years
14% 9–12 years
27–50% >12 years
70% athletes
- Normal: <50 PVCs/24 hrs

Reference

Kugler, JD. Benign arrhythmias: neonate throughout childhood. In Deal BJ, Wolff GS, Gelband, H, eds, *Current Concepts in Diagnosis and Management of Arrhythmia in Infants and Children*. Armonk, NY: Futura Publishing, 1998, 81–83.

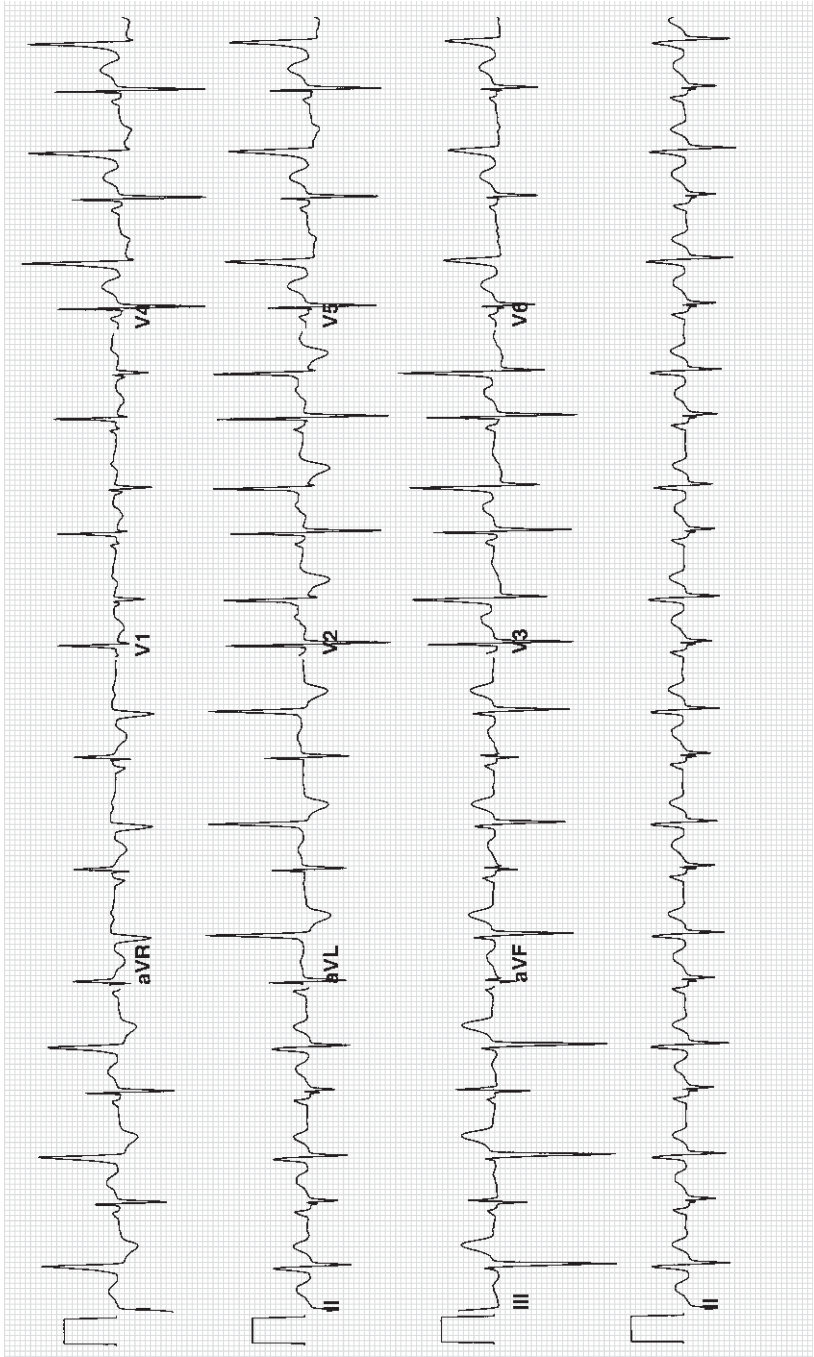


Figure 95 1-week-old infant with irregular heart rhythm.

Figure 95

- Sinus rhythm with frequent premature ventricular complexes
- Right atrial enlargement
Rightward QRS axis ($+200^\circ$) at upper limits of normal for age

Ventricular bigeminy

- Premature ventricular complexes alternate with sinus beats

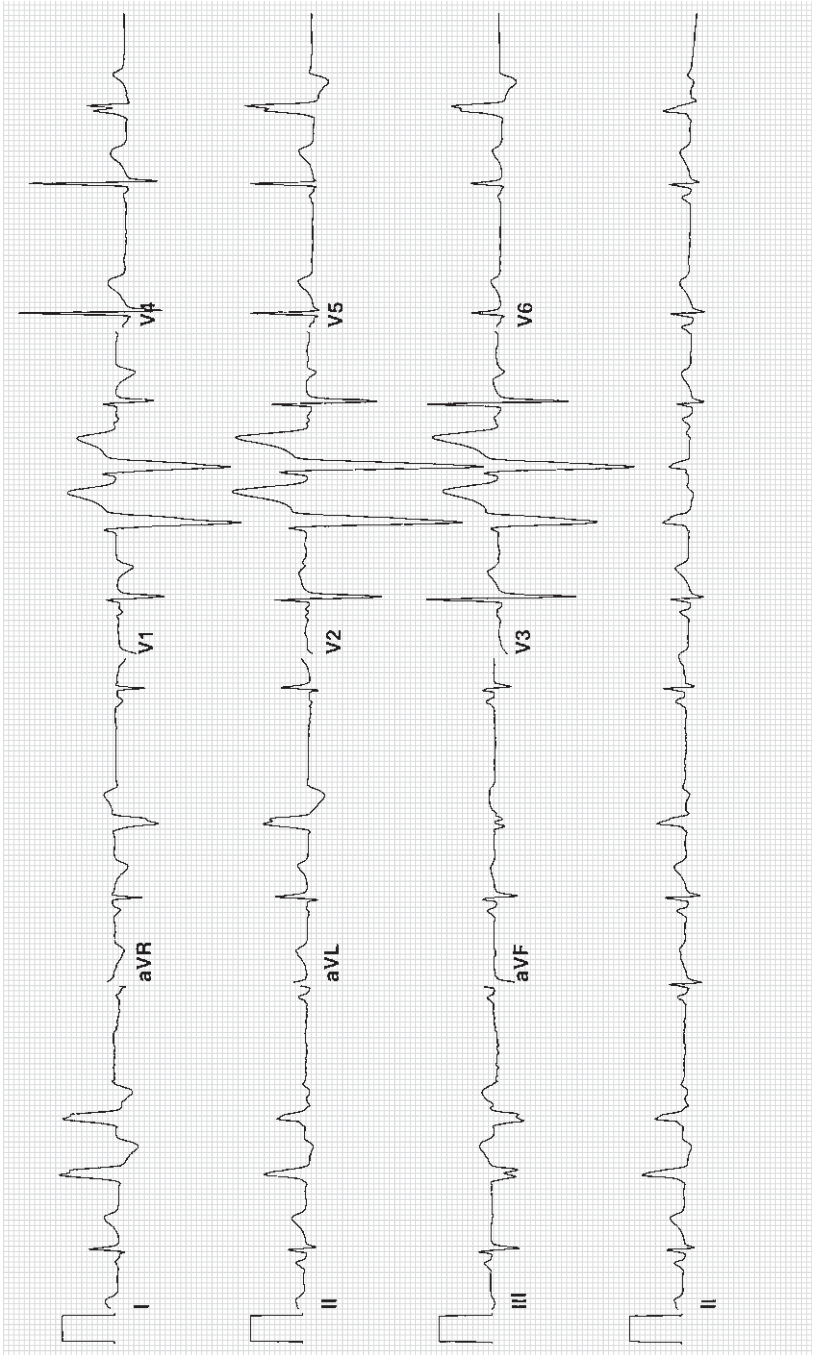


Figure 96 8-year-old girl evaluated for chest pain.

Figure 96

- Sinus rhythm, 90 bpm
- Frequent uniform ventricular couplets (consecutive PVCs)
- PVC morphology:
 - Left bundle branch pattern suggests origin from right ventricle
 - Superior axis deviation suggests origin from apex
- Voltages and intervals of sinus beats are normal

Ventricular couplets

- Paired premature ventricular contractions
- Uncommon in children with normal hearts

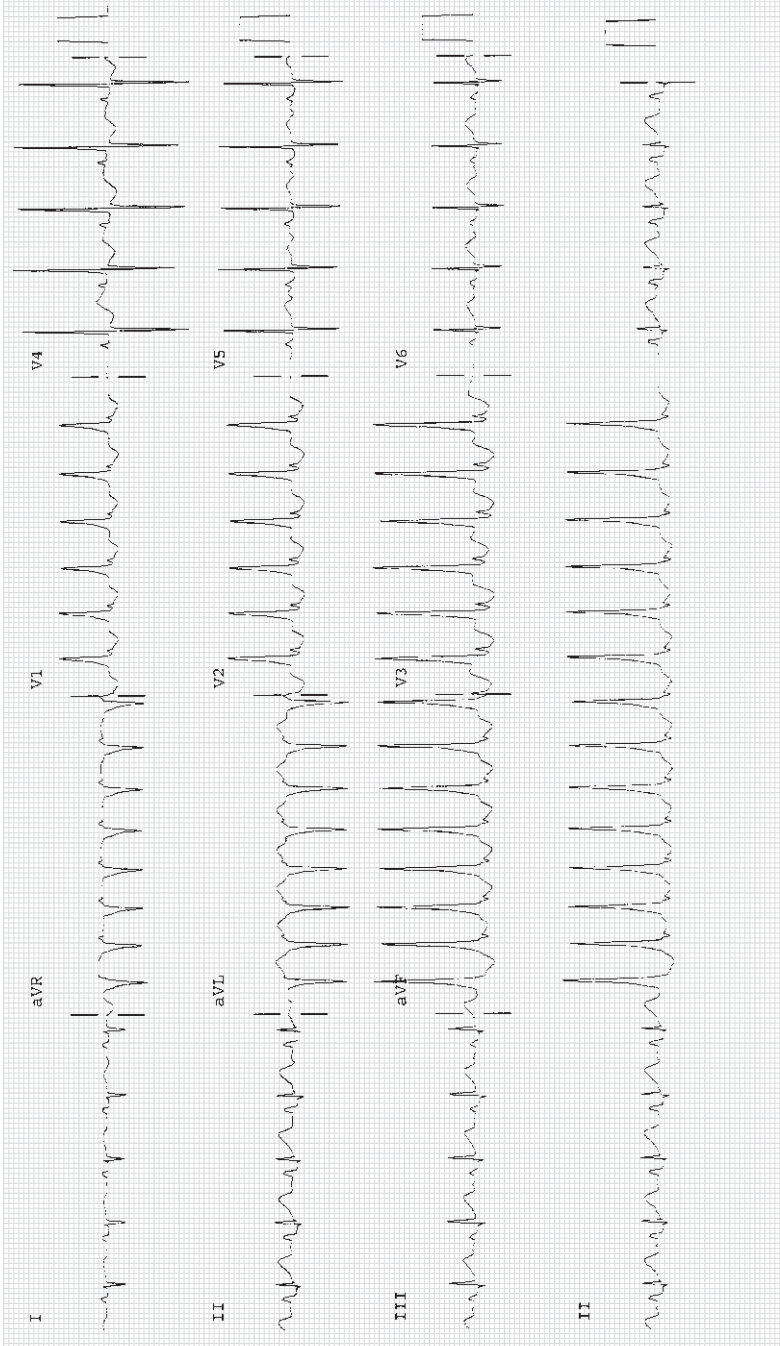


Figure 97 Neonate with irregular rhythm. (ECG courtesy of Andrew Griffin, M.D.)

Figure 97

- Sinus rhythm at 120 bpm, with runs of wide QRS rhythm at 155–175 bpm
- Retrograde P wave follows wide QRS complexes in all ventricular beats except first, indicating ventriculoatrial (VA) conduction
- Normal intervals and voltages of sinus conducted beats

Accelerated ventricular rhythm

- Ventricular rhythm with rate slightly faster than sinus rate (usually <10–20% faster)
- QRS duration prolonged
- Usually seen in infants or less commonly in older children, with structurally normal hearts
- Typically does not require treatment, and spontaneously resolves over months to years

Reference

MacLellan-Tobert SG, Porter CJ. Accelerated idioventricular rhythm: A benign arrhythmia in childhood. *Pediatrics* 1995;**96**:122–125.

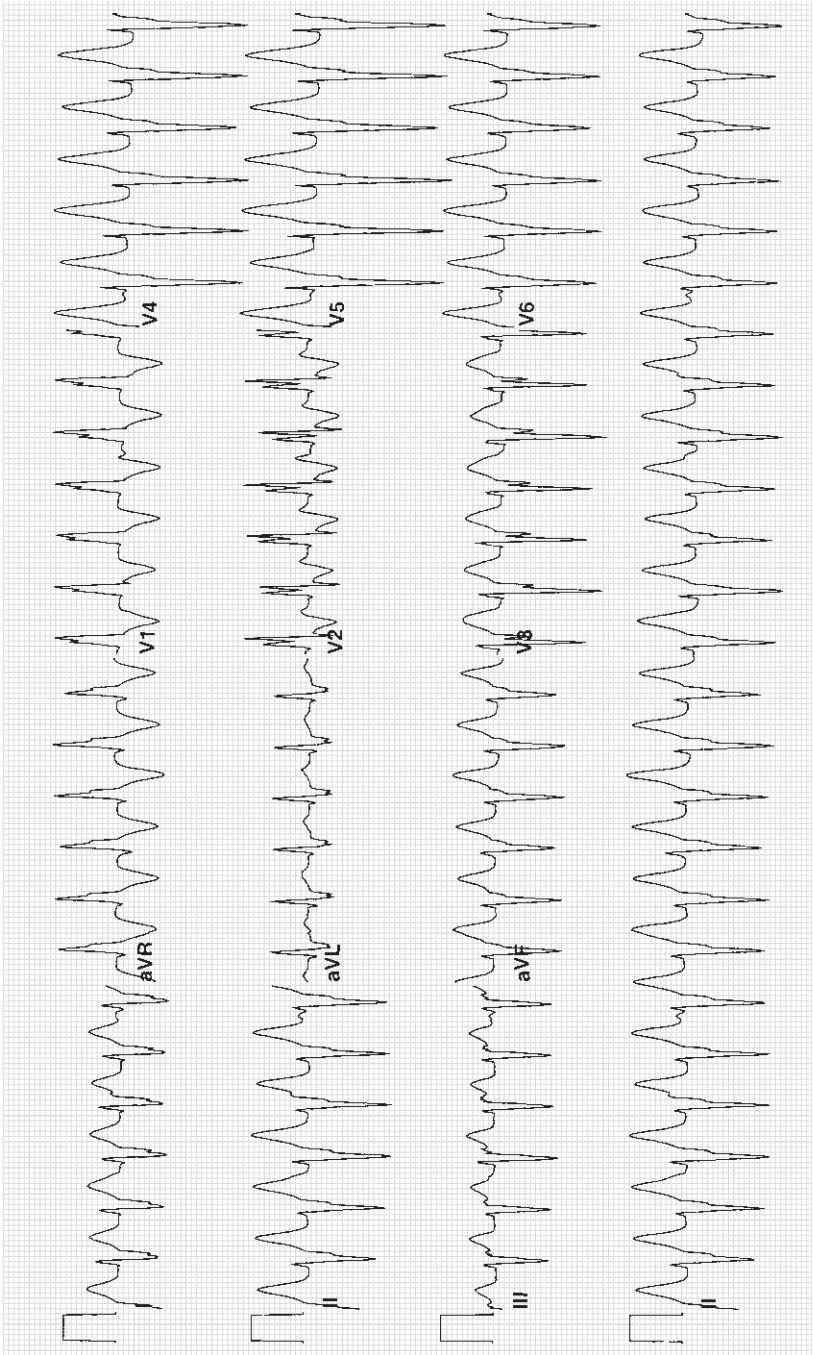


Figure 98 11-year-old boy with exertional tachycardia.

Figure 98

- Regular wide complex tachycardia, 150 bpm
- QRS duration = 0.130 sec
- QRS morphology:
 - Right bundle branch block pattern suggests origin from left ventricle
 - Superior QRS axis suggests inferior origin
- Atrioventricular dissociation, best seen in leads I, II

Idiopathic left ventricular tachycardia

- Wide complex tachycardia of right bundle branch block, left axis deviation morphology
- Originates from mid- to apical septal surface of left ventricle
- Usually occurs in adolescents with structurally normal hearts
- Often verapamil-sensitive

Reference

Belhassen B, Shapira I, Pelleg A, Copperman I, Kauli N, Laniado S. Idiopathic recurrent sustained ventricular tachycardia responsive to verapamil: an ECG-electrophysiologic entity. *Am Heart J* 1984;**108**:1034–1037.

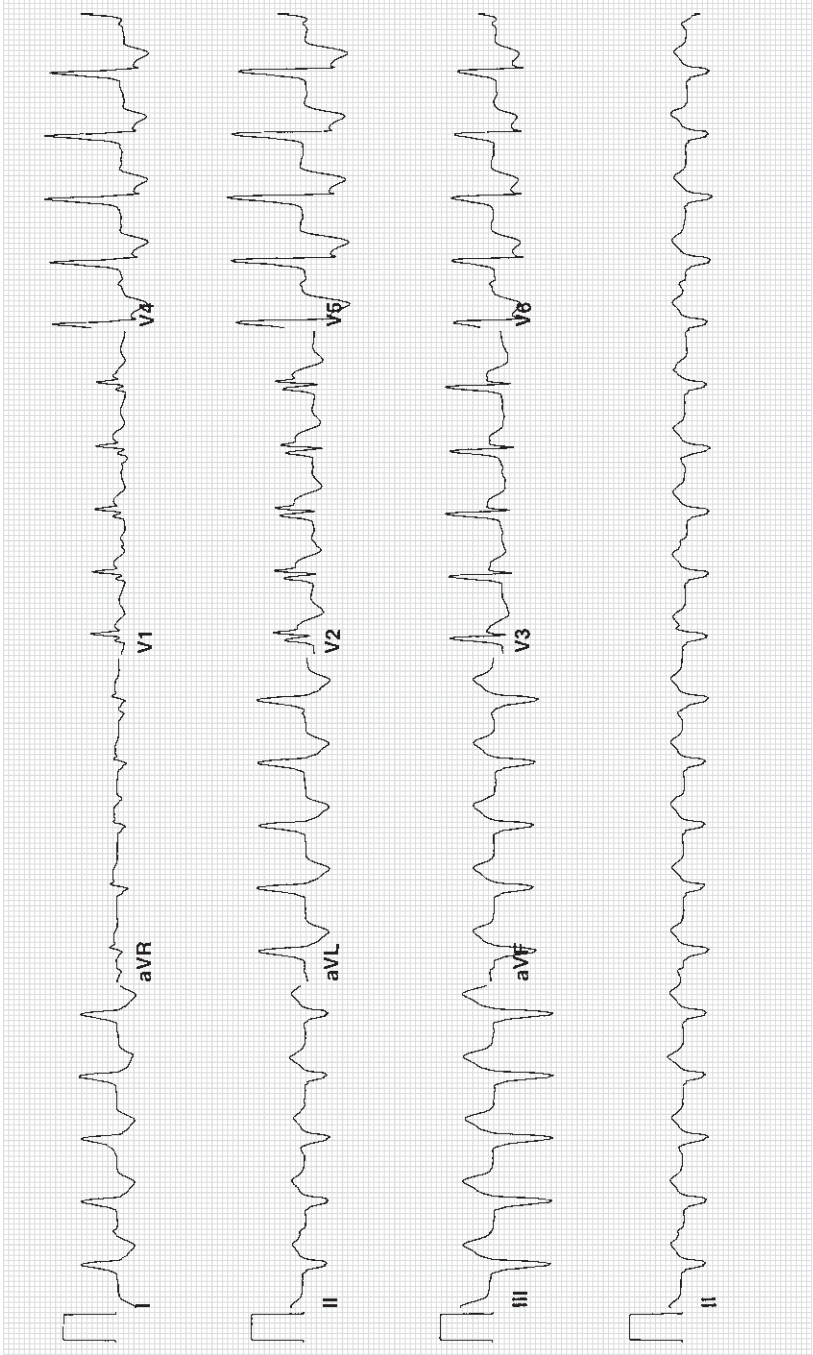


Figure 99 10-year-old boy with 3-day history of fever, vomiting, and malaise.

Figure 99

- Regular wide QRS rhythm, 125bpm
- QRS duration = 0.120 sec
- Atrioventricular dissociation, with P waves best seen in leads II, V1, V6
- QRS morphology:
 - Right bundle branch block suggests origin from left ventricle
 - Superior QRS axis suggests origin from apex
- Abnormal ST changes in chest leads

Ventricular tachycardia (VT)

In patients with previously normal hearts, VT may be associated with:

- Electrolyte or metabolic abnormalities: hyper- or hypokalemia, hypercalcemia, hypomagnesemia, acidosis, hypoxemia, hypoglycemia
- Sympathomimetic agents
- Halothane anesthesia
- Infection: myocarditis, endocarditis
- Primary cardiac electrical disease:
 - long QT syndrome, Brugada syndrome, catecholaminergic tachycardia

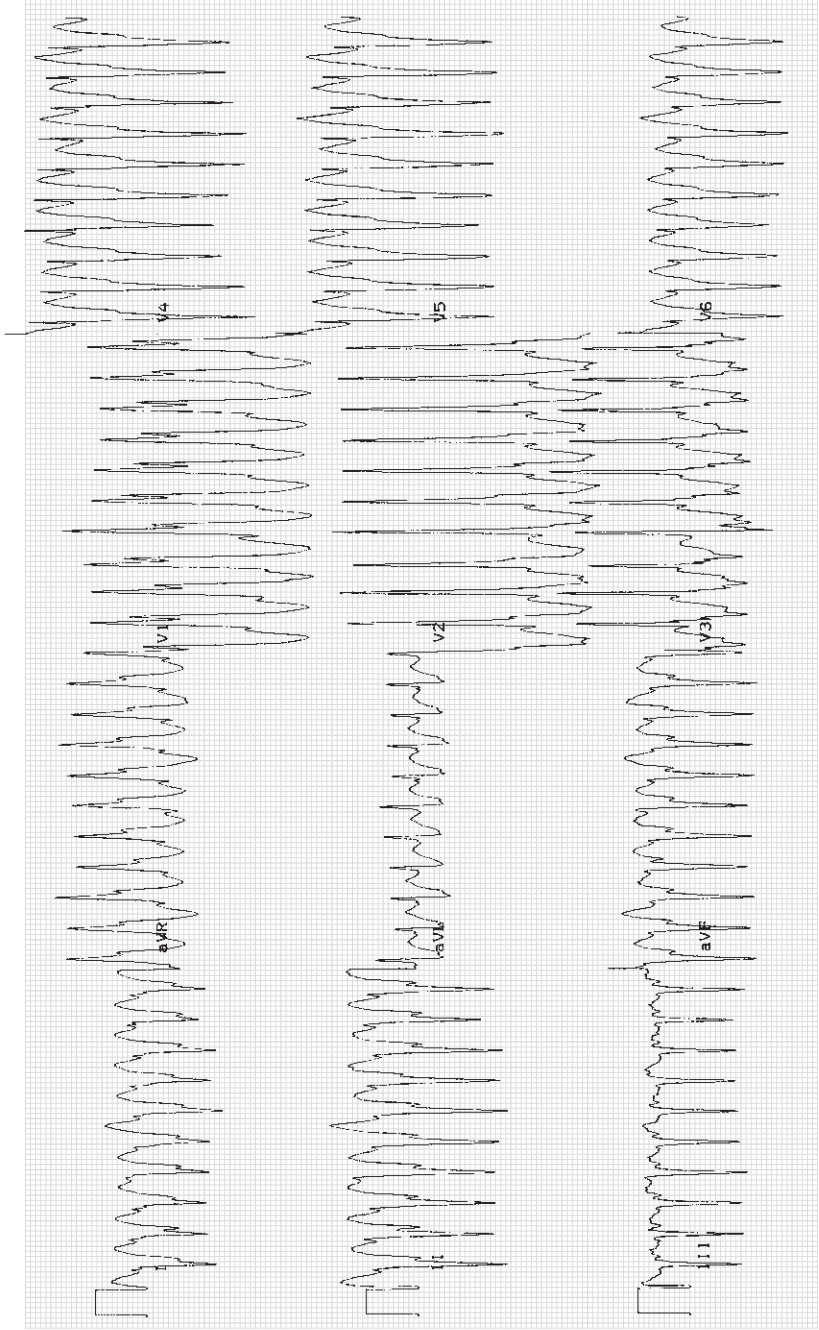


Figure 100 1-year-old boy with fever and hemodynamic collapse.

Figure 100

- Regular wide QRS tachycardia, 250 bpm
- QRS duration = 0.180 sec
- Difficult to discern P waves
- QRS morphology:
 - Right bundle branch block suggests left ventricular origin
 - Superior QRS axis suggests origin from apex

Wide QRS tachycardia

- Criteria favoring VT:
- VA dissociation
 - QRS duration >0.015 sec longer than 98th percentile for age
 - Superior QRS axis
 - Fusion beats

Reference

Tipple MA. Usefulness of the electrocardiogram in diagnosing mechanisms of tachycardia. *Pediatr Cardiol* 2000;**21**:516–521.

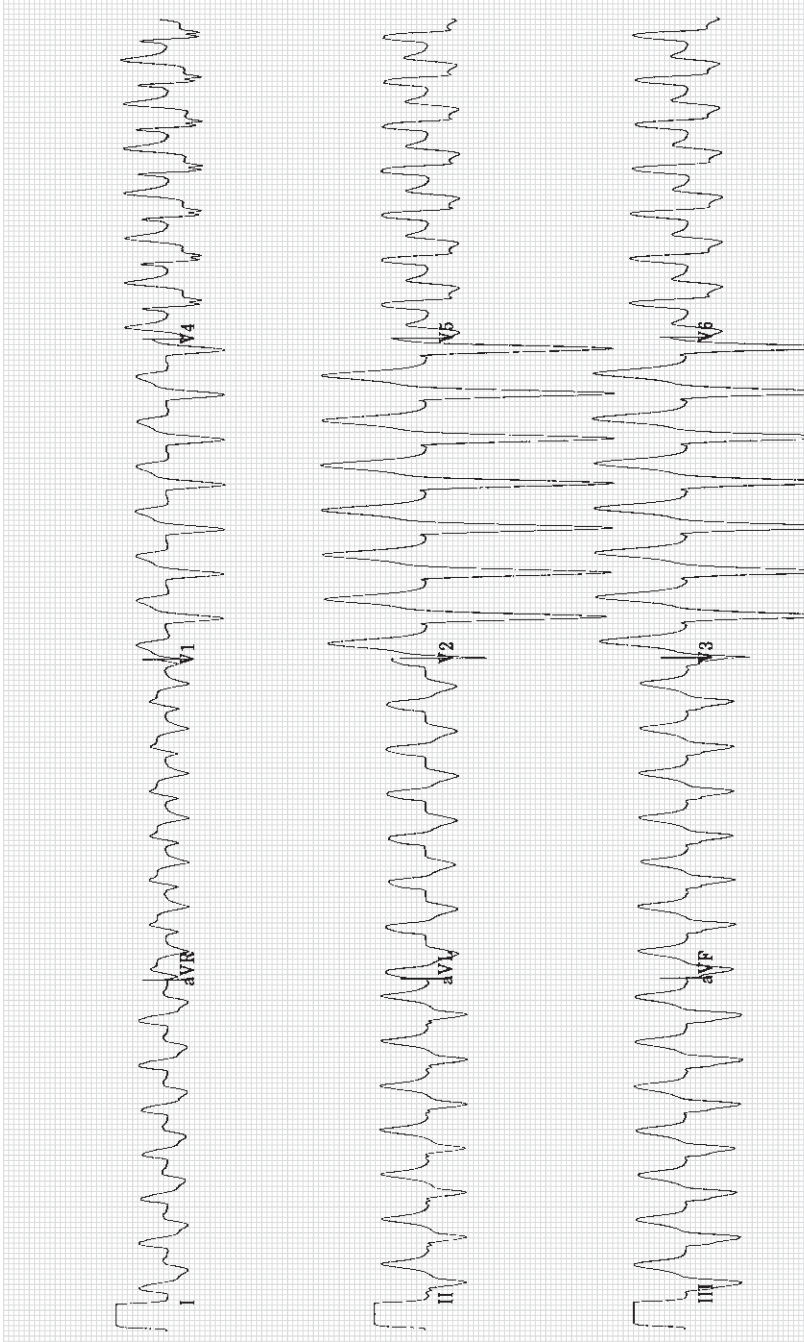


Figure 101 6-year-old boy, 6 hours post-intracardiac repair of aortic stenosis; tachycardia responded to removal of mediastinal chest tube.

Figure 101

- Regular wide QRS tachycardia, 170 bpm
- QRS duration = 0.125 sec
- P waves not visible
- QRS morphology:

Left bundle branch block suggests origin from right ventricle

Superior QRS axis suggests origin from ventricular apex

Wide complex tachycardia: VT versus SVT

Criteria favoring VT in the presence of a left bundle branch block morphology:

- Any Q wave in lead V6
- R wave in V1 or V2 >0.03 sec
- Onset of R wave to nadir of S wave duration >0.06 sec in V1 or V2
- Notching on the downstroke of the S wave in V1 or V2

References

- Antunes E, Brugada J, Steurer G, Andries E, Brugada P. The differential diagnosis of a regular tachycardia with a wide QRS complex on the 12 lead ECG. *PACE* 1994;17:1515–1522.
- Brugada P, Brugada J, Mont L, Smeets J, Andries EW. A new approach to the differential diagnosis of a regular tachycardia with a wide QRS complex. *Circulation* 1991;83:1649–1659.

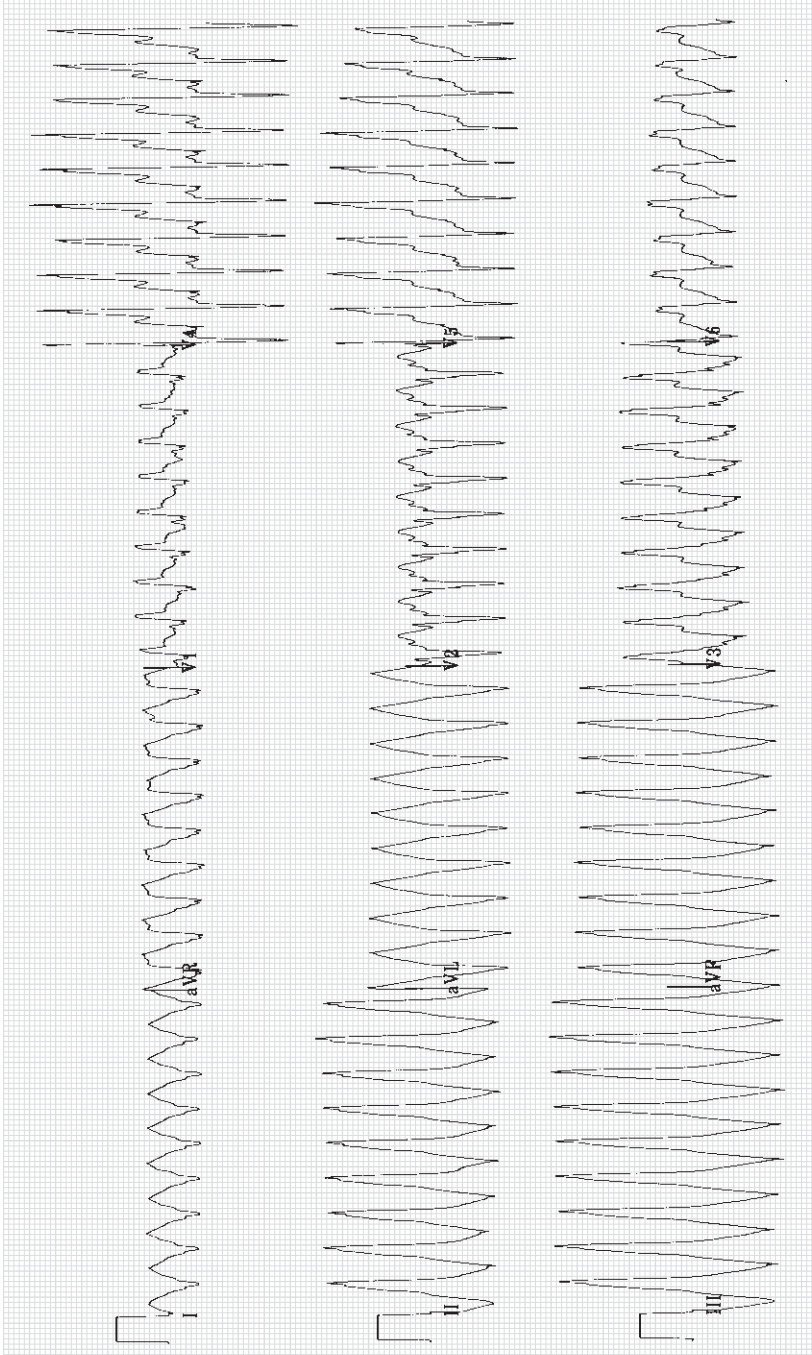


Figure 102 8-year-old boy, status post-repair of double outlet right ventricle: syncope with exertion.

Figure 102

- Regular wide QRS tachycardia, 220 bpm
- QRS duration = 0.16 sec
- P waves difficult to discern
- QRS morphology:
 - Right bundle branch block morphology suggests left ventricular origin
 - Inferior QRS axis suggests origin from outflow tract

Wide complex tachycardia: VT versus SVT

Criteria favoring ventricular tachycardia in presence of right bundle branch block morphology:

- Left axis deviation
- Triphasic pattern of QRS complex
- R/S ratio <1 in lead V6

References

- Antunes E, Brugada J, Steurer G, Andries E, Brugada P. The differential diagnosis of a regular tachycardia with a wide QRS complex on the 12 lead ECG. *PACE* 1994;17:1515–1522.
- Brugada P, Brugada J, Mont L, Smeets J, Andries EW. A new approach to the differential diagnosis of a regular tachycardia with a wide QRS complex. *Circulation* 1991;83:1649–1659.

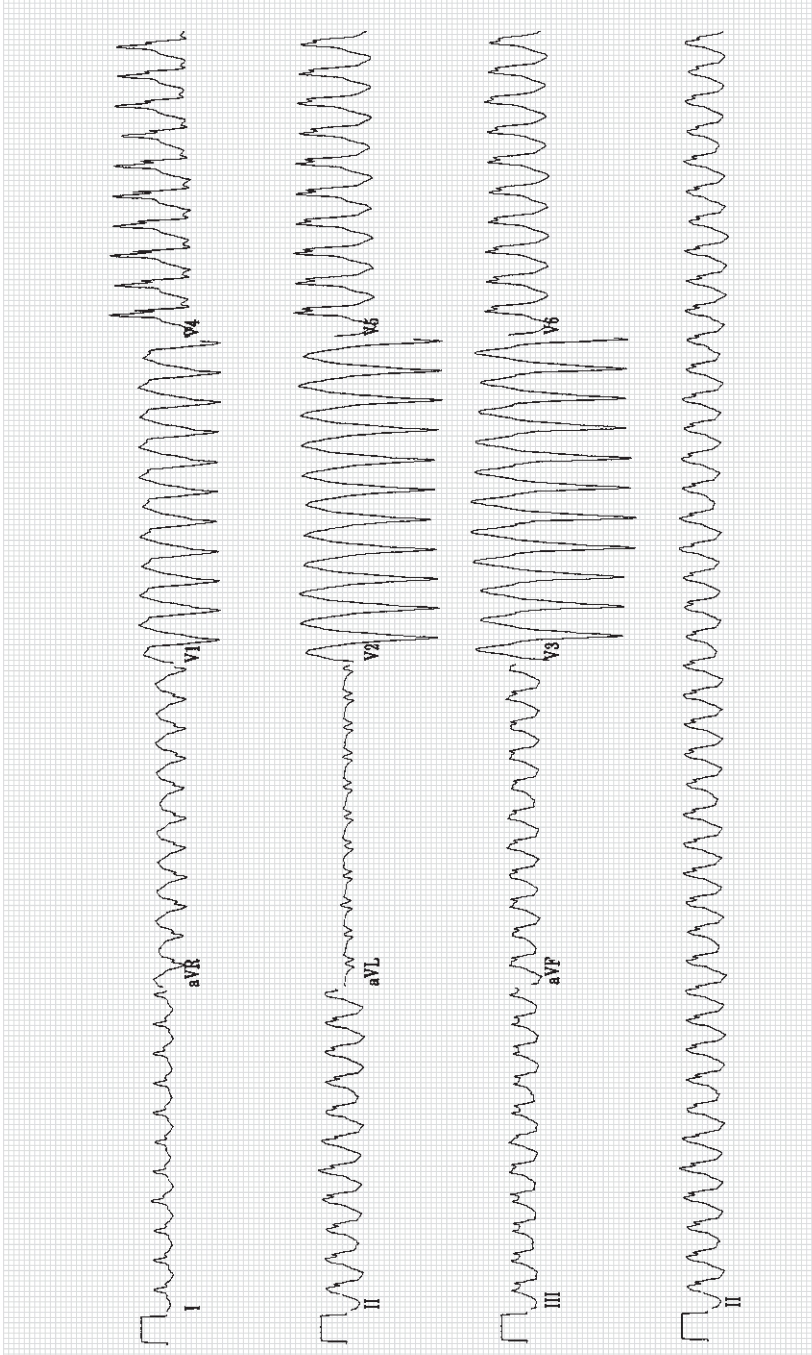


Figure 103 4-year-old boy with recurrent tachycardia, status post-repair of double outlet right ventricle. (ECG courtesy of Ernerio Alboliras, M.D.)

Figure 103

- Regular wide QRS tachycardia, 260 bpm
- P waves not easily seen; difficult to identify AV relationship
- QRS duration = 0.15 sec
- QRS morphology:
 - Left bundle branch block pattern suggests origin from right ventricle
 - Inferior axis (+60°) suggests origin from outflow tract
- R onset to nadir of S wave in V1 >0.06 sec suggests ventricular tachycardia

Ventricular tachycardia

Sustained VT in patients with congenital heart disease:

- May be life-threatening
- Requires aggressive treatment to convert acute episode and prevent recurrences: medication, ablation, surgery, or implantable defibrillator

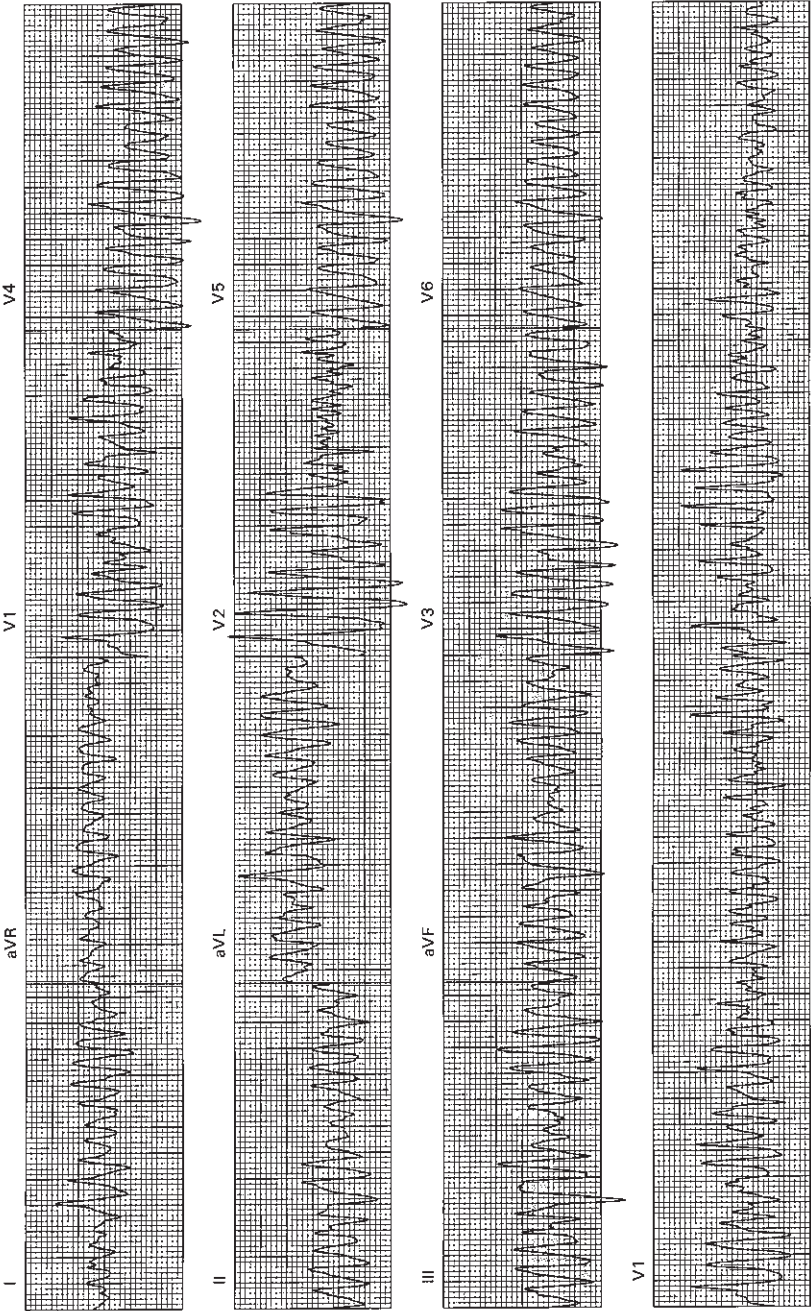


Figure 104 5-year-old boy with cardiac arrest.

Figure 104

- Irregular, polymorphic wide QRS tachycardia, rate >300 bpm

Ventricular fibrillation

- Irregularly irregular very rapid ventricular activity, rates 150–500 bpm
- Chaotic appearance of QRS complexes
Lethal arrhythmia, requires prompt defibrillation

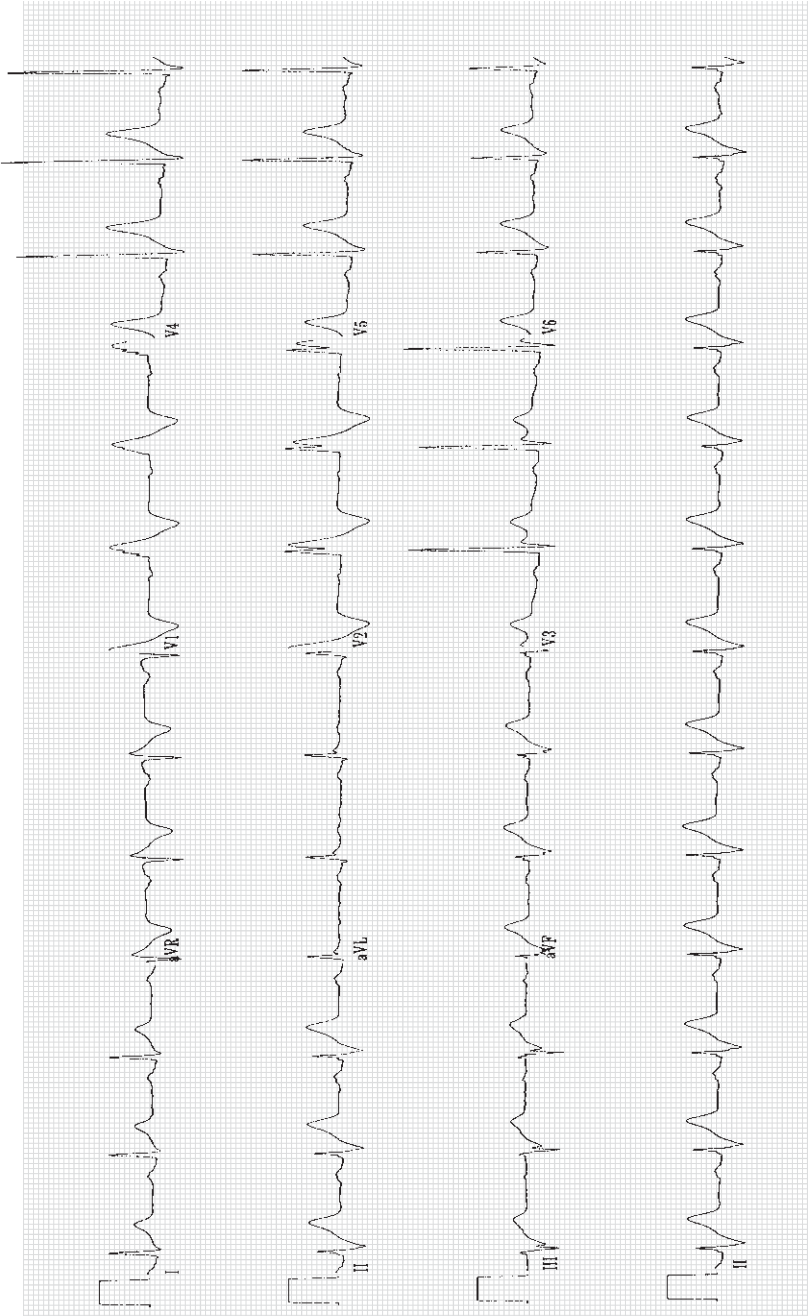


Figure 105 30-year-old male with strong family history of sudden death. (ECG courtesy of David Ruschhaupt, M.D.)

Figure 105

- Normal sinus rhythm, 76bpm
- Right bundle branch block pattern
- Elevated ST segments, best seen in leads V1–V3

Brugada Syndrome

- ECG findings: right bundle branch block, ST segment elevation in leads V1–V3
- Absence of structural heart disease
- Very rare form of familial primary cardiac electrical disease
- High incidence of sudden cardiac death
- Differential diagnosis:
 - Right ventricular dysplasia
 - Myocardial ischemia, injury
 - Tricyclic anti-depressant toxicity
 - Mediastinal tumor

Reference

Marcus FI. Electrocardiographic features of inherited diseases that predispose to the development of cardiac arrhythmias, long QT syndrome, arrhythmogenic right ventricular cardiomyopathy/dysplasia, and Brugada syndrome. *J Electrocardiol* 2000;33(Suppl.):1–10. Review.

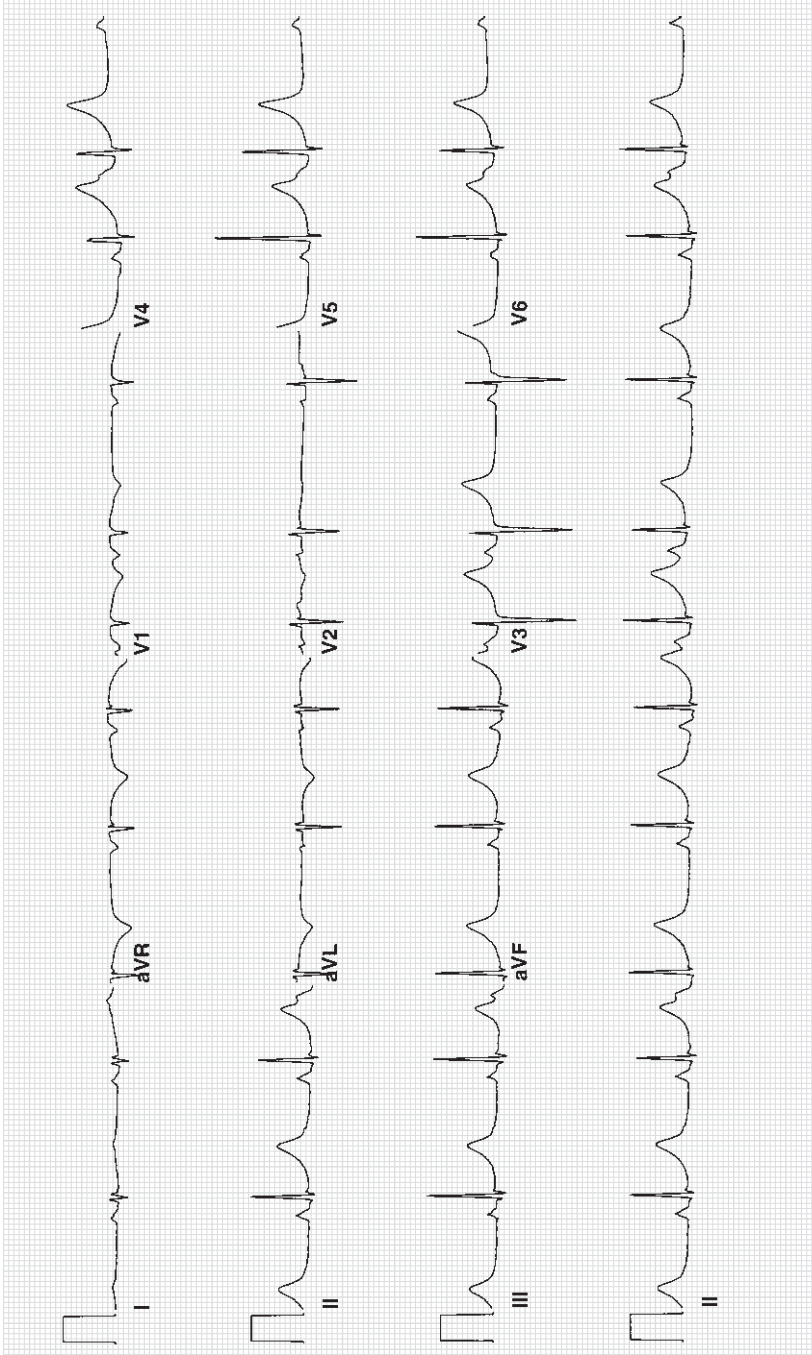


Figure 106 12-year-old girl with exertional syncope; history of “seizures.”

Figure 106

- Sinus rhythm with sinus arrhythmia, 67 bpm
- Marked QT prolongation, $QT_c = 0.62$ sec
- Abnormal repolarization pattern of T wave:
Broad based T wave
T wave extends to P wave in leads V4–V6

Long QT Syndrome (LQTS)

ECG findings:

- Prolonged corrected QT interval and abnormal T wave morphologies in most patients
- QT interval must be corrected for heart rate
Measure QT and RR interval in seconds
Bazett's formula for QT correction
 $QT_c = \text{measured QT interval divided by square root of RR interval}$
- Normal QT_c :
 - <0.46 females
 - <0.45 males
 - <0.48 newborn infants

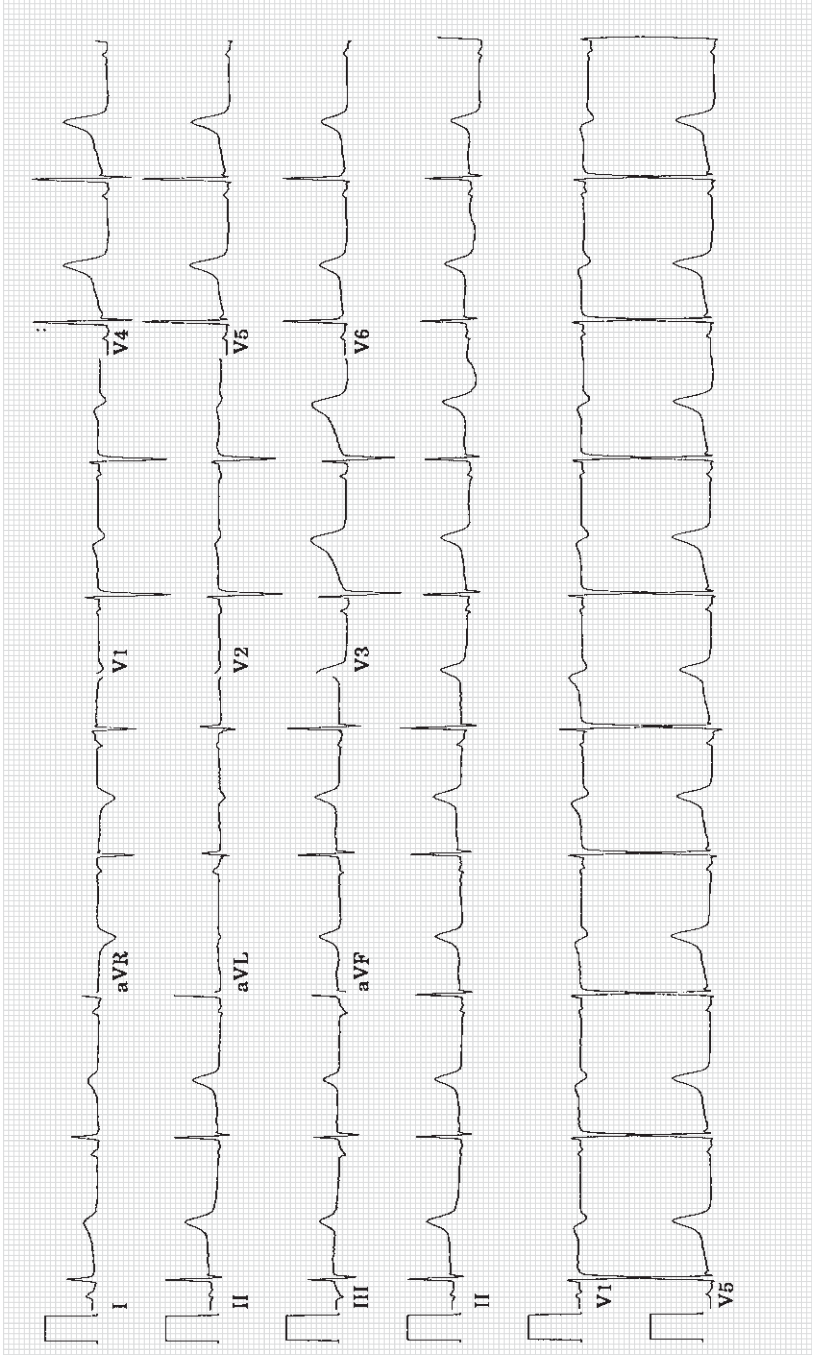


Figure 107 10-year-old boy with syncope. (ECG courtesy of Michael Ackerman, M.D.)

Figure 107 (12-15-96)

- Sinus bradycardia, 55 bpm
- Prolonged QT interval, QTc = 0.51 sec
- Abnormal repolarization pattern of T wave:
Long isoelectric ST segment
Prominent late peaking T wave

Prolonged QT interval

- Congenital long QT syndrome: familial primary electrical disease due to abnormal potassium or sodium channels
- Acquired form: secondary to electrolyte or metabolic derangement in susceptible patients
- Increased risk of ventricular arrhythmias, particularly *torsades de pointes*
- High risk of syncope, seizures, or sudden death

References

- Ackerman MJ. The long QT syndrome: ion channel diseases of the heart. *Mayo Clin Proc* 1998;**73**(3):250–259.
- Vincent GM. The molecular genetics of the long QT syndrome: genes causing fainting and sudden death. *Annu Rev Med* 1998;**49**:263–274.

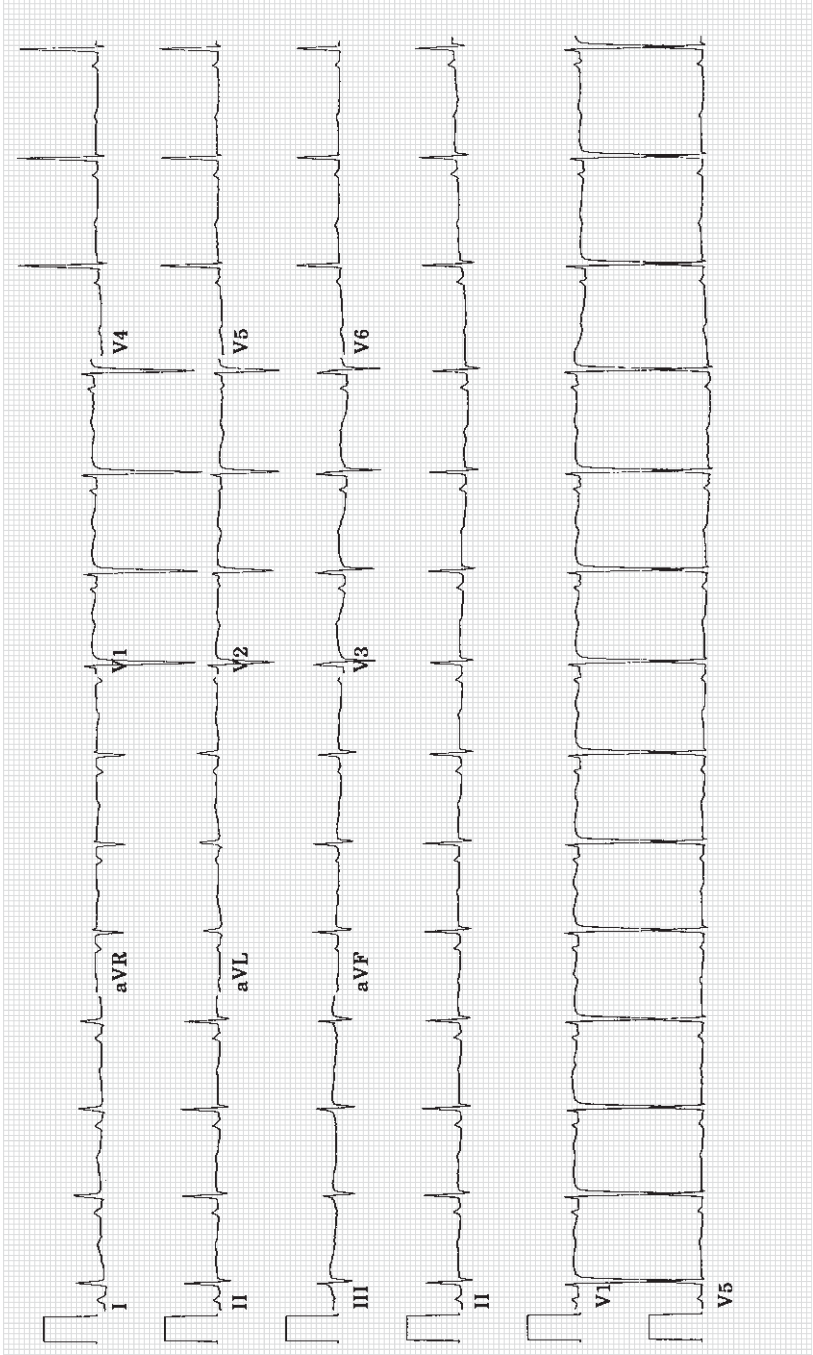


Figure 108 10-year-old boy with syncope, same patient as prior ECG. (ECG courtesy of Michael Ackerman, M.D.)

Figure 108 (12-6-96)

- Sinus rhythm, 80 bpm
- QTc at upper limits of normal
- Abnormal repolarization pattern of T wave
 - Low amplitude, flattened T waves
- Note QT interval and T wave morphology differences compared to prior tracing

Long QT syndrome

- Some LQTS patients have only mildly prolonged or normal QTc at times
- T wave morphology and QTc may vary significantly over time in same patient

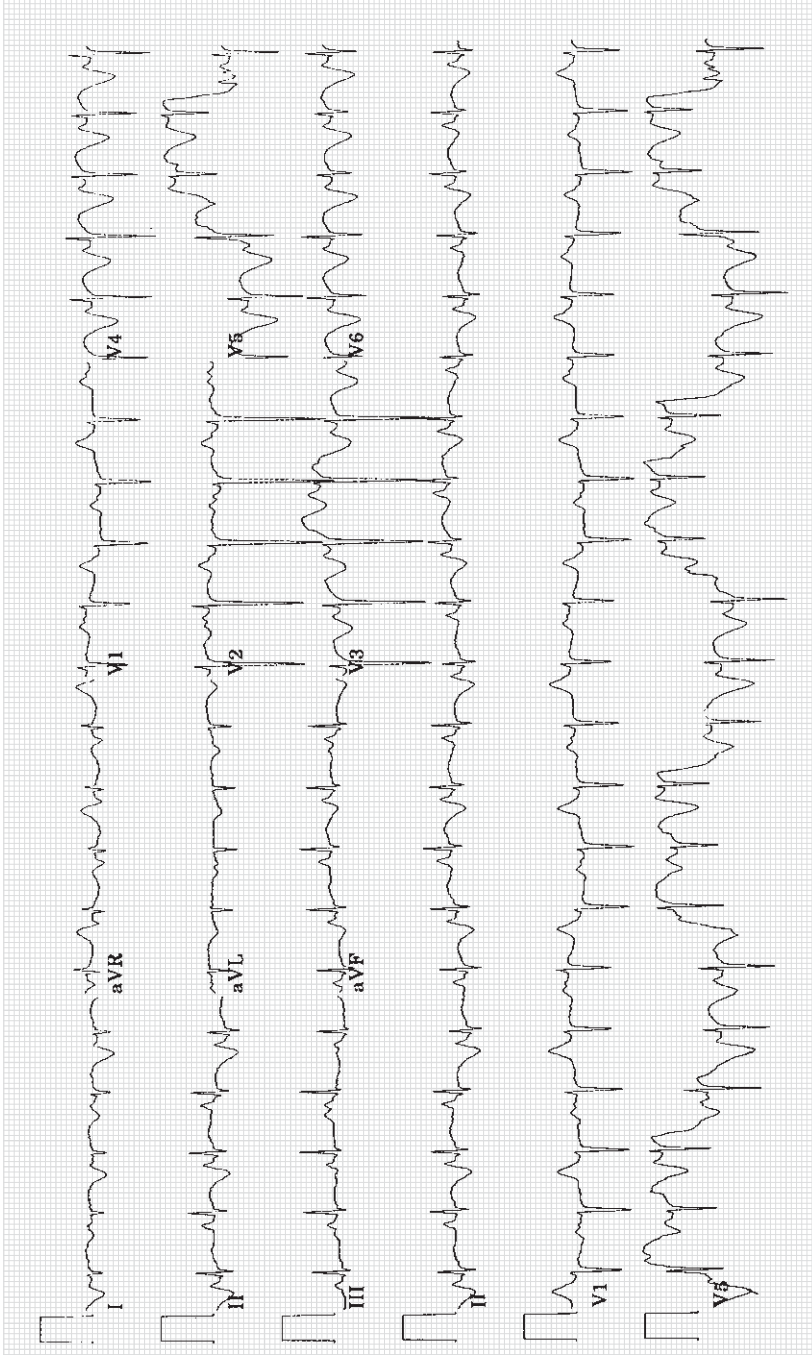


Figure 109 19-year-old male with near-drowning episode. (ECG courtesy of Michael Ackerman, M.D.)

Figure 109

- Sinus tachycardia, 124 bpm
- Marked QT prolongation, $QT_c = 0.56$ sec
- Poor R wave progression in V2–V6
- T wave inversion in lateral leads
- T wave alternans best seen in leads II and V1

Long QT Syndrome

- T wave alternans:
Variation in T wave amplitude and/or axis on alternate beats
- Ominous finding associated with high risk of imminent ventricular tachycardia

Reference

Franklin WH, Hickey RW. Images in clinical medicine. Long-QT syndrome. *N Engl J Med* 1995;**333**:355.

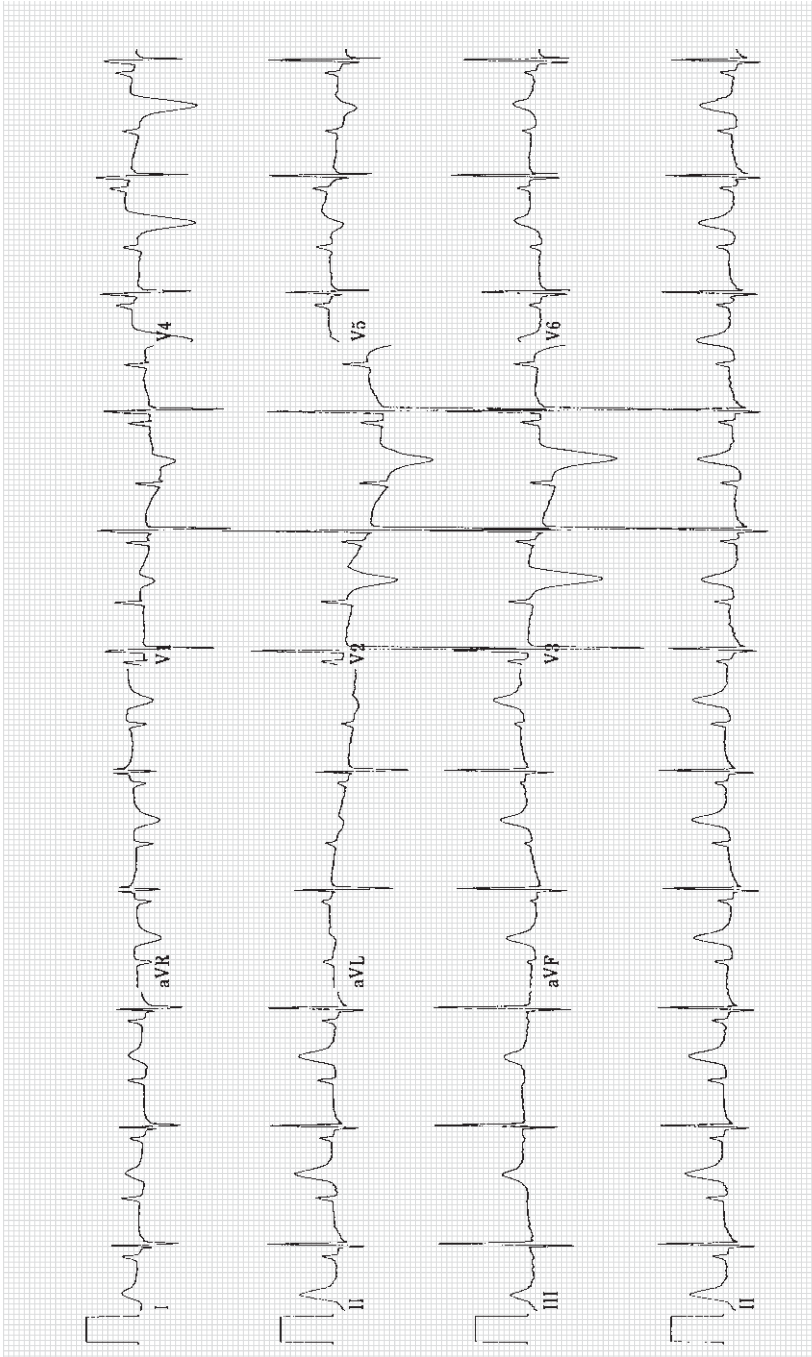


Figure 110 Neonate with bradycardia and seizures. (ECG courtesy of John Hokanson, M.D.)

Figure 110

- Sinus rhythm, atrial rate 130 bpm
- 2:1 atrioventricular conduction:
 Ventricular rate = 65 bpm
 Every other P wave falls within ST segment of preceding beat
- Marked QTc prolongation, $QT_c = 0.71$ sec:
 Prolonged isoelectric ST segment
 Late onset of T wave
- Right atrial enlargement
- Left ventricular hypertrophy

Long QT Syndrome

- Sinus bradycardia or atrioventricular block (particularly in neonates) may be associated with LQTS

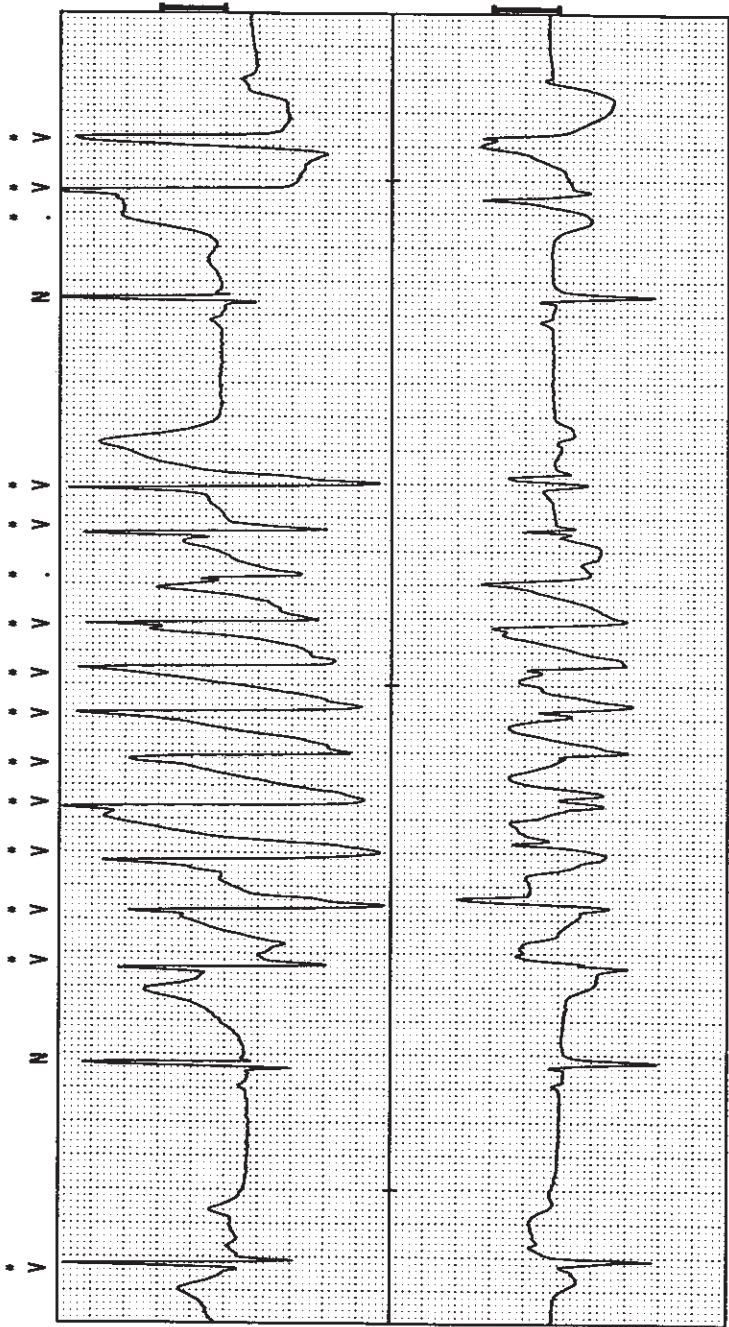


Figure 111 Holter: 7-year-old boy with “seizure disorder.”

Figure 111

Holter:

- Sinus bradycardia, followed by nonsustained polymorphic ventricular tachycardia (*torsades de pointes*)
- *Torsades de pointes* onset with “long-short” coupling sequence: bradycardia followed by premature ventricular complex triggers VT
- Bizarre T waves of sinus beats with QT prolongation

Torsades de pointes

- Polymorphic ventricular tachycardia characterized by alternating QRS polarity (“twisting of points”)
- Rate >250bpm
- Occurs in setting of prolonged QT interval
- *Torsades de pointes* may be nonsustained, causing dizziness or syncope, or sustained and degenerating to ventricular fibrillation

Reference

Passman R, Kadish A. Polymorphic ventricular tachycardia, long Q-T syndrome, and torsades de pointes. *Med Clin North Am* 2001;**85**(2):321–341.

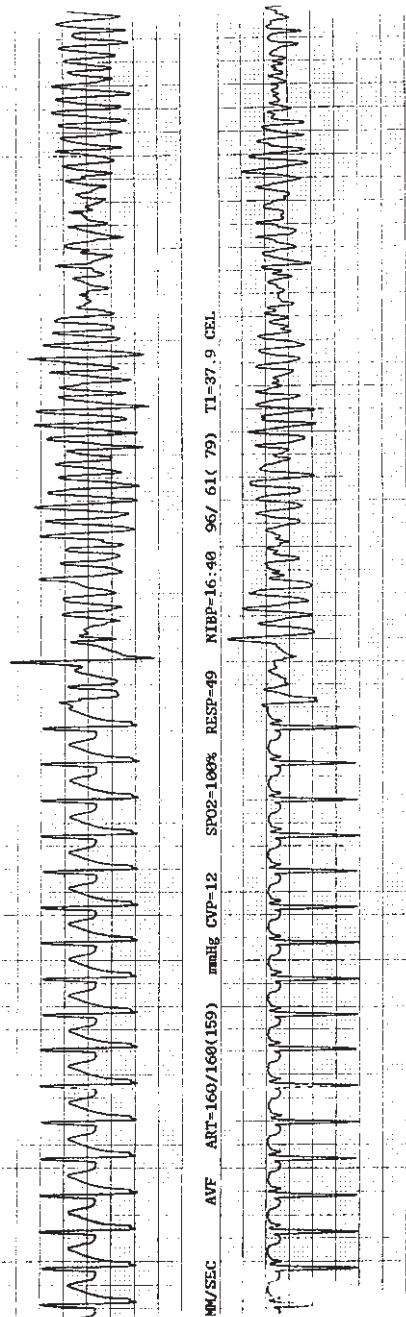


Figure 112 Holter: 7-year-old boy with "seizure disorder."

Figure 112

Rhythm strip:

- Longer episode of *torsades de pointes*

Torsades de pointes

- Polymorphic ventricular tachycardia characterized by alternating QRS polarity (“twisting of points”)
- Rate >250bpm
- Occurs in setting of prolonged QT interval
- *Torsades de pointes* may be nonsustained, causing dizziness or syncope, or sustained and degenerating to ventricular fibrillation

Reference

Passman R, Kadish A. Polymorphic ventricular tachycardia, long Q-T syndrome, and torsades de pointes. *Med Clin North Am* 2001;85(2):321–341.

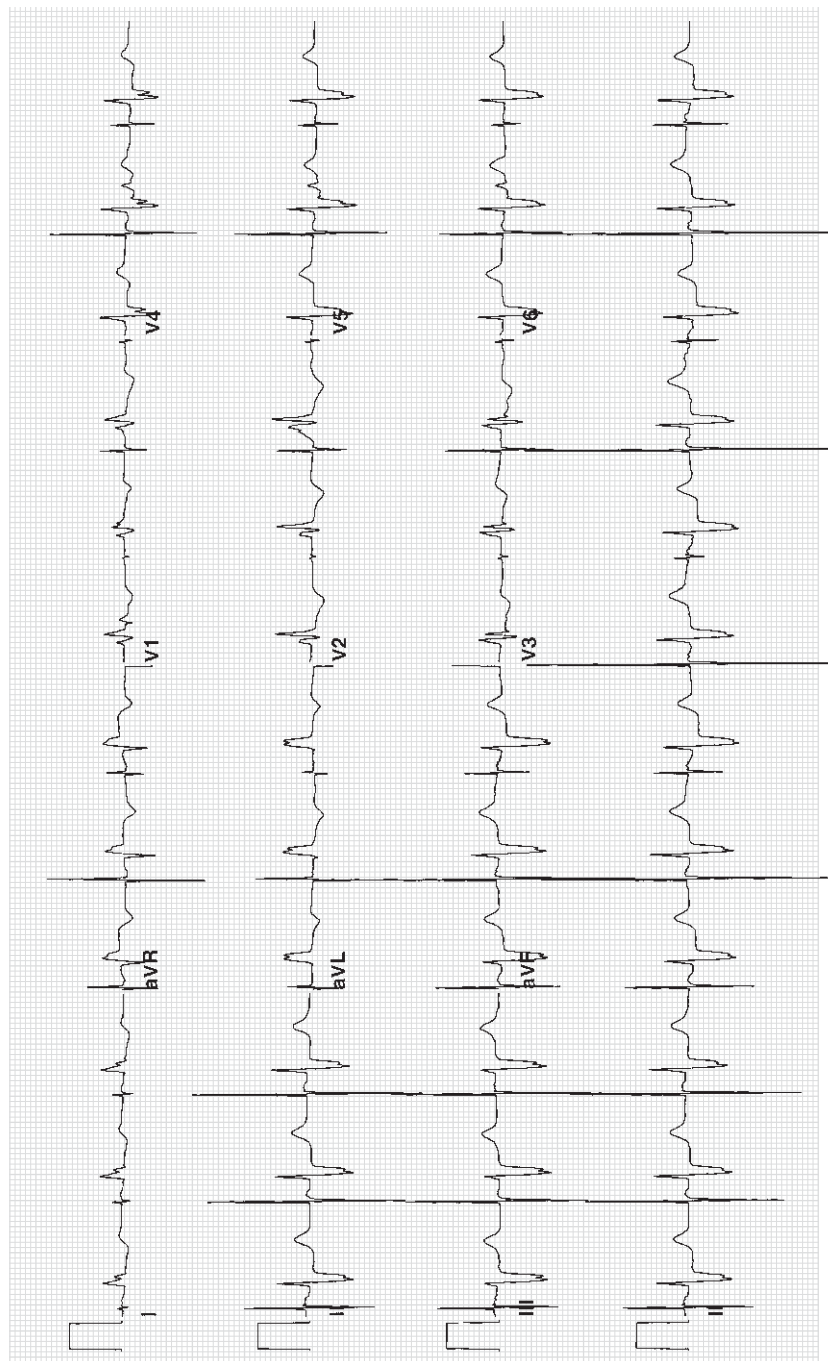


Figure 113 15-year-old girl with single-chamber pacemaker.

Figure 113

- Tall, sharp pacing artifact immediately followed by P wave, 70 bpm
- Normal atrioventricular conduction
- Left axis deviation
- Right bundle branch block
- Note nonconducted premature atrial contraction in leads V4–V6

AAI pacing*

- Atrial lead provides atrial sensing, with programmable atrial pacing rate
- When intrinsic atrial rate falls below programmed lower rate limit, atrial pacing occurs
- When intrinsic atrial rate exceeds programmed lower rate limit, atrial pacer is inhibited from delivering pacing impulse
- Atrial pacing used for patients with significant sinus bradycardia and intact AV nodal function
- Provides anti-bradycardia pacing support

*See p. 12 for pacing nomenclature.

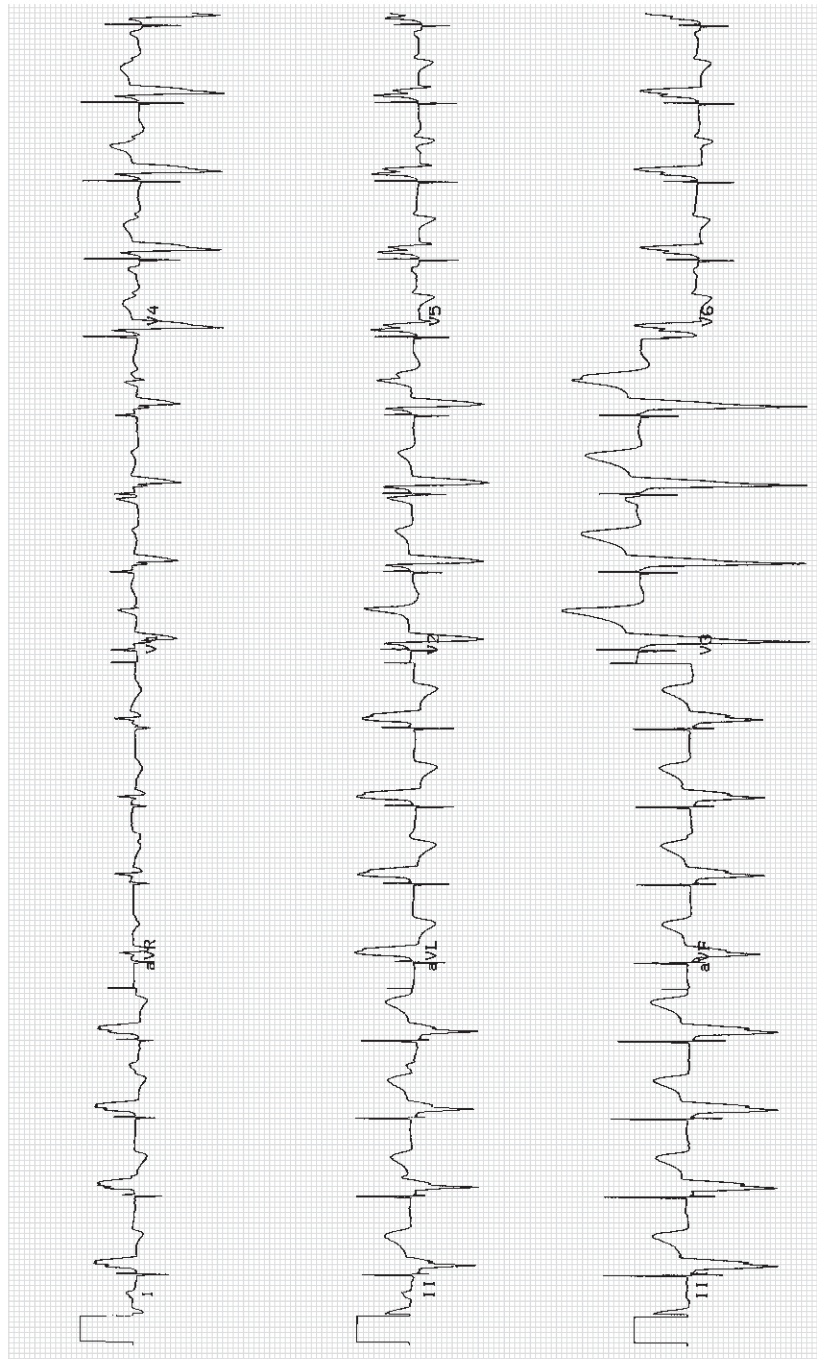


Figure 114 1-year-old boy with congenital complete heart block; ventricular pacemaker.

Figure 114

- Complete atrioventricular block:
Sinus rate 68 bpm (best seen in leads II, V1, V4)
Ventricular rate 105 bpm
- Sharp pacing artifact immediately precedes each QRS complex
- Right atrial enlargement
- QRS morphology: left bundle branch block pattern, superior axis indicates pacing from right ventricular apex

VVI Pacing*

- Ventricular lead provides ventricular sensing, with programmable ventricular pacing rate
- When intrinsic ventricular rate falls below programmed lower rate, ventricular pacing occurs
- When intrinsic ventricular rate exceeds programmed lower rate limit, ventricular pacing inhibited from delivering pacing impulse
- VVI pacing does not provide for atrioventricular synchrony
- Ventricular pacing indicated for patients with significant bradycardia and atrioventricular block
- Criteria for ventricular hypertrophy or intrinsic bundle branch block do not apply to ventricular paced beats

*See p. 12 for pacing nomenclature.

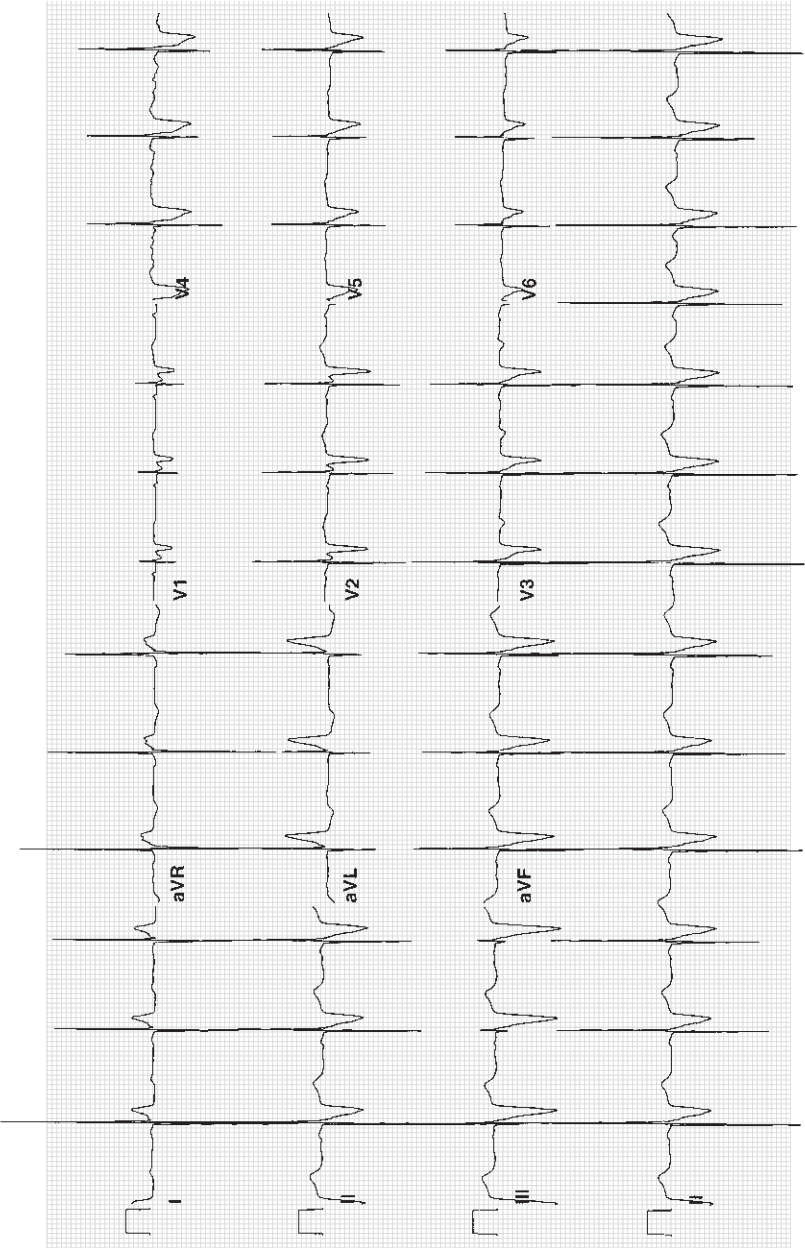


Figure 115 16-year-old girl with dual-chamber pacemaker for complete heart block.

Figure 115

- P waves followed by pacing spike, followed by wide QRS complexes
- Atrial tracking:
 - Sinus beats are sensed, followed by programmed atrioventricular delay, and ventricular pacing
- QRS morphology:
 - Left bundle branch block pattern, left axis deviation due to pacing from right ventricular apex

DDD Pacing

- Atrial and ventricular leads provide dual chamber sensing and pacing
- Sensing of both chambers, with inhibition of either atrial or ventricular pacing when intrinsic activity detected at rate > lower rate limit detected
- Programmable lower and upper pacing rate limits
- Programmable atrioventricular delay
- DDD pacing provides atrioventricular synchrony, and allows intrinsic sinus rhythm to determine paced ventricular rate

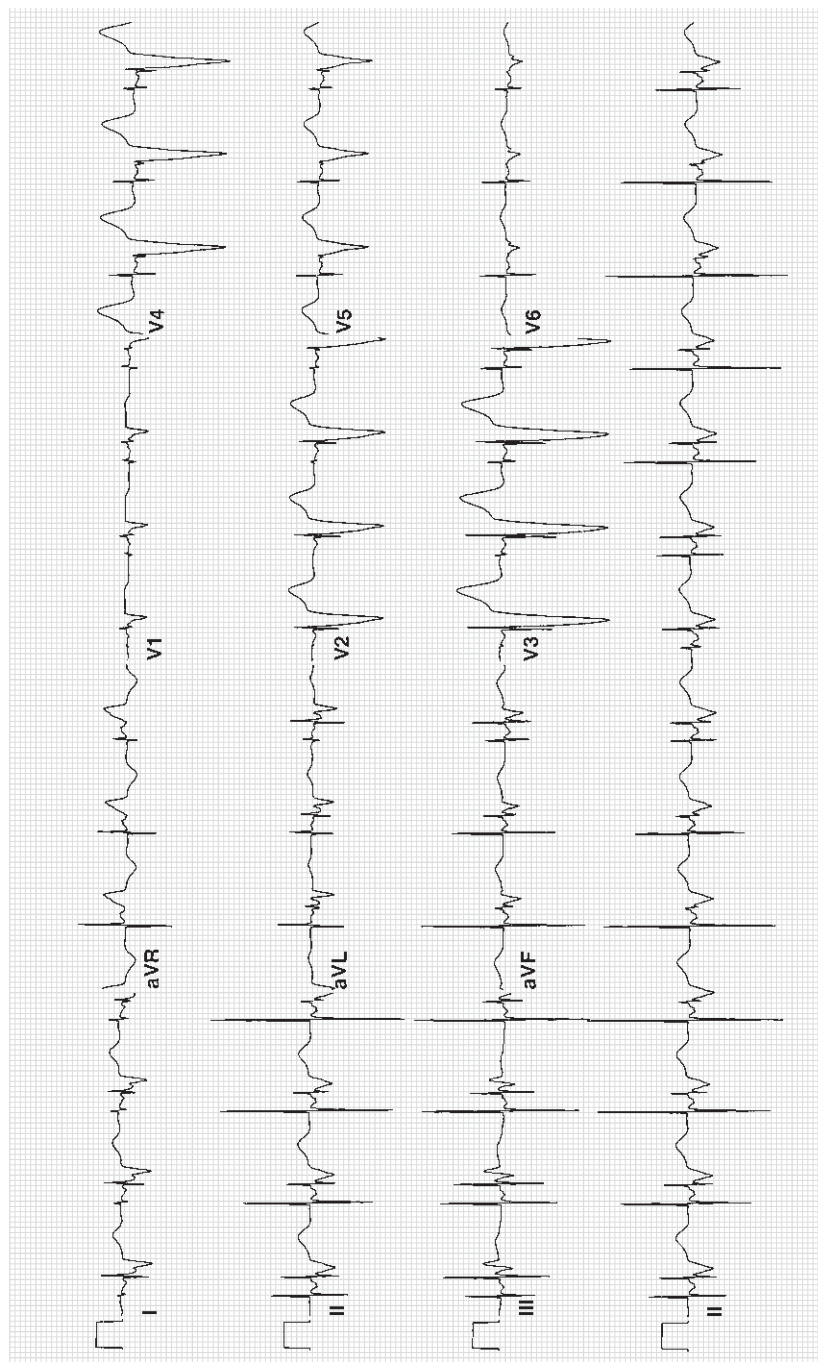


Figure 116 13-year-old female with dual-chamber pacemaker.

Figure 116

- Atrial and ventricular pacing spikes are followed by P waves and QRS complexes, respectively (atrioventricular sequential pacing)
- Heart rate 85 bpm
- Atrioventricular delay = 0.14 sec
- QRS morphology:
Left bundle branch block, left axis deviation due to pacing from right ventricular apex

DDD pacing

- Atrioventricular sequential pacing:
Both atria and ventricles are paced, with programmed atrioventricular delay (paced PR interval)
- Programmable lower and upper heart rate limits and atrioventricular delay (PR interval)
- When intrinsic sinus rate is lower than programmed lower rate limit, the atrium is paced:
If the programmed atrioventricular delay elapses and the atrial impulse has not conducted to the ventricle, the ventricle is paced
- DDD pacing is useful for patients with significant sinus node dysfunction and atrioventricular block

*See p. 12 for pacing nomenclature.

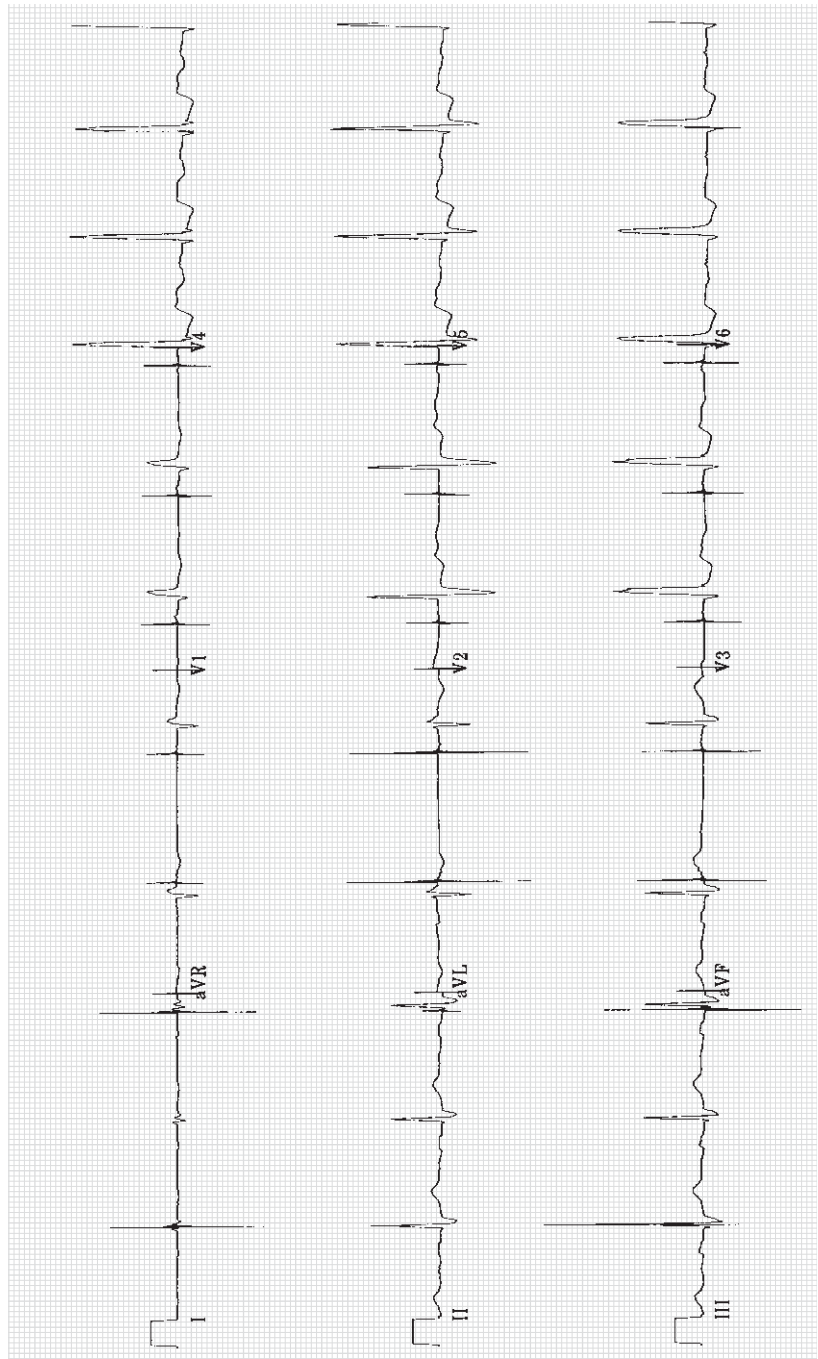


Figure 117 18-year-old male, status post-Mustard procedure for transposition of the great arteries, with atrial pacemaker.

Figure 117

- Rhythm alternates between sinus (low amplitude P waves) and atrial pacing
- First three and last two atrial pacing spikes occur despite immediately preceding intrinsic sinus beats, suggesting intrinsic atrial rhythm was “undersensed” intermittently
- The 2nd and 9th sinus beats are appropriately sensed by the pacemaker
- Intact AV conduction
- Right ventricular hypertrophy
- T wave inversion in lateral leads

Pacemaker Malfunction: Undersensing

- Pacemaker malfunction may be due to either sensing or capture abnormalities
- Undersensing: pacemaker does not appropriately detect underlying rhythm, resulting in inappropriate pacing
- Atrial undersensing: atrial pacing despite intrinsic P waves immediately preceding the pacer spikes
- Inappropriate pacing may result in irregular rhythm or induce tachycardia

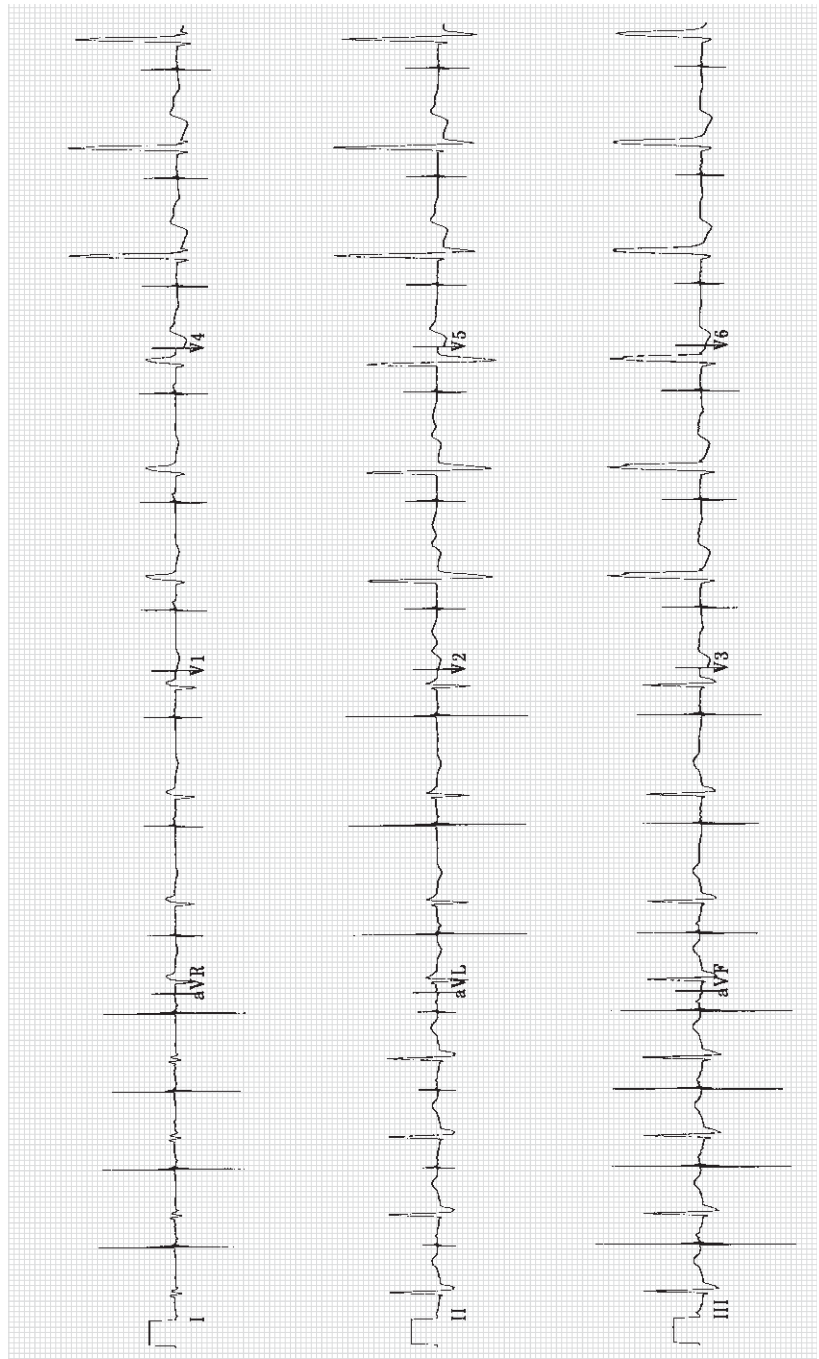


Figure 118 18-year-old male, status post-Mustard procedure for transposition of the great arteries, with atrial pacemaker.

Figure 118

- Paced atrial rhythm, at 100 bpm, abruptly dropping to 73 bpm
- Intact AV conduction
- Right ventricular hypertrophy
- T wave inversion in lateral leads

Pacemaker Malfunction: Oversensing

- Atrial oversensing occurs when nonatrial activity (e.g. ventricular activity) is sensed as an intrinsic atrial beat
- Oversensing may cause inappropriate inhibition of pacing, and reset the pacemaker clock
- Oversensing may result in bradycardia

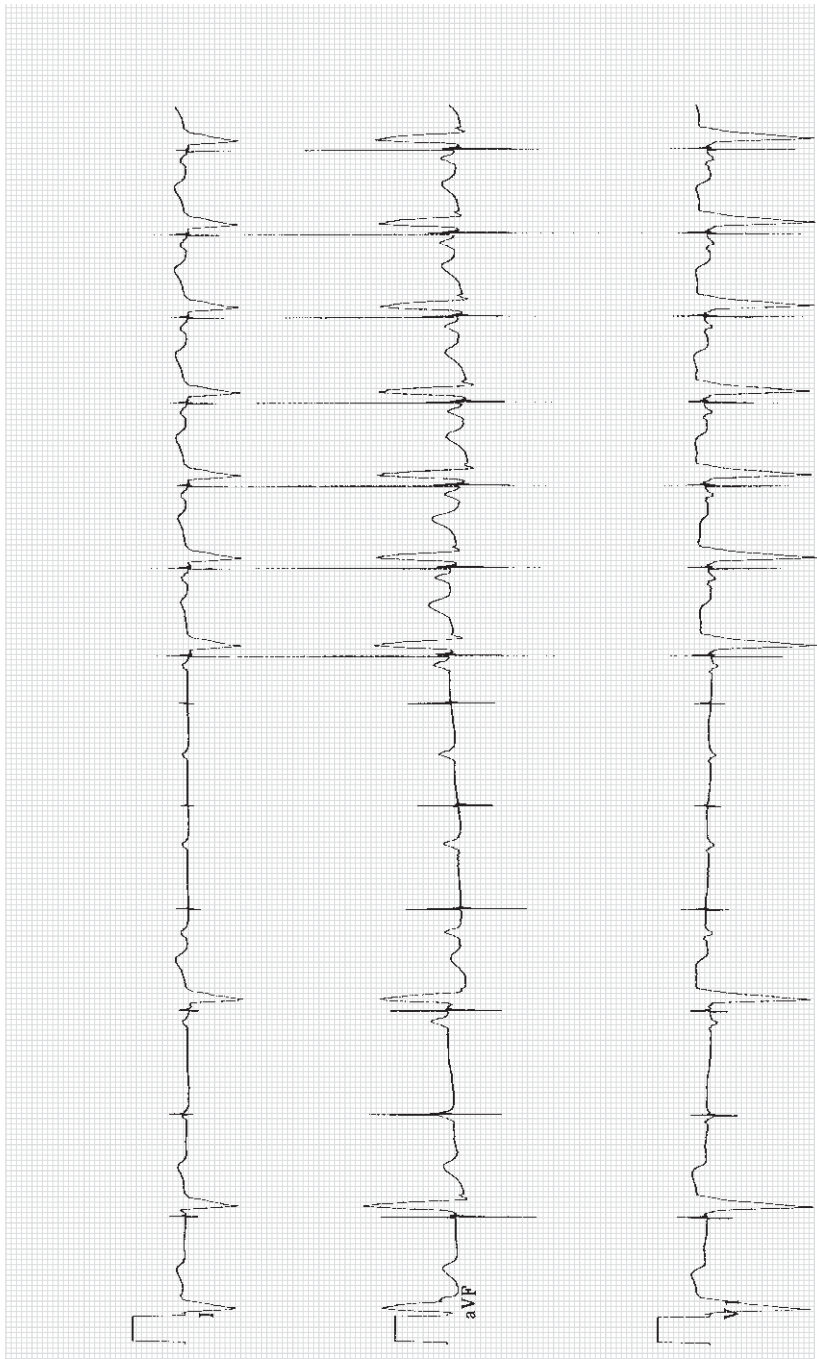


Figure 119 11-year-old with surgical complete heart block; dual-chamber pacemaker.

Figure 119

- Two ventricular paced beats, followed by pacing artifact falling inside P wave. Fourth beat shows P wave followed by pacing artifact with ventricular capture.
- Pacing artifacts then fire without ventricular capture. Note intrinsic P waves at 90 bpm
- Last seven beats show atrial tracking with appropriate ventricular capture (P wave followed by pacing spike, with ventricular complex immediately following)

Pacemaker Malfunction: Failure to capture

- Pacemaker noncapture: appropriately timed pacing spike delivered, but no subsequent P wave (atrial noncapture) or QRS complex (ventricular noncapture)
- Pacemaker sensing and capture functions occur independently
- Failure to capture the ventricle may result in bradycardia, and symptoms

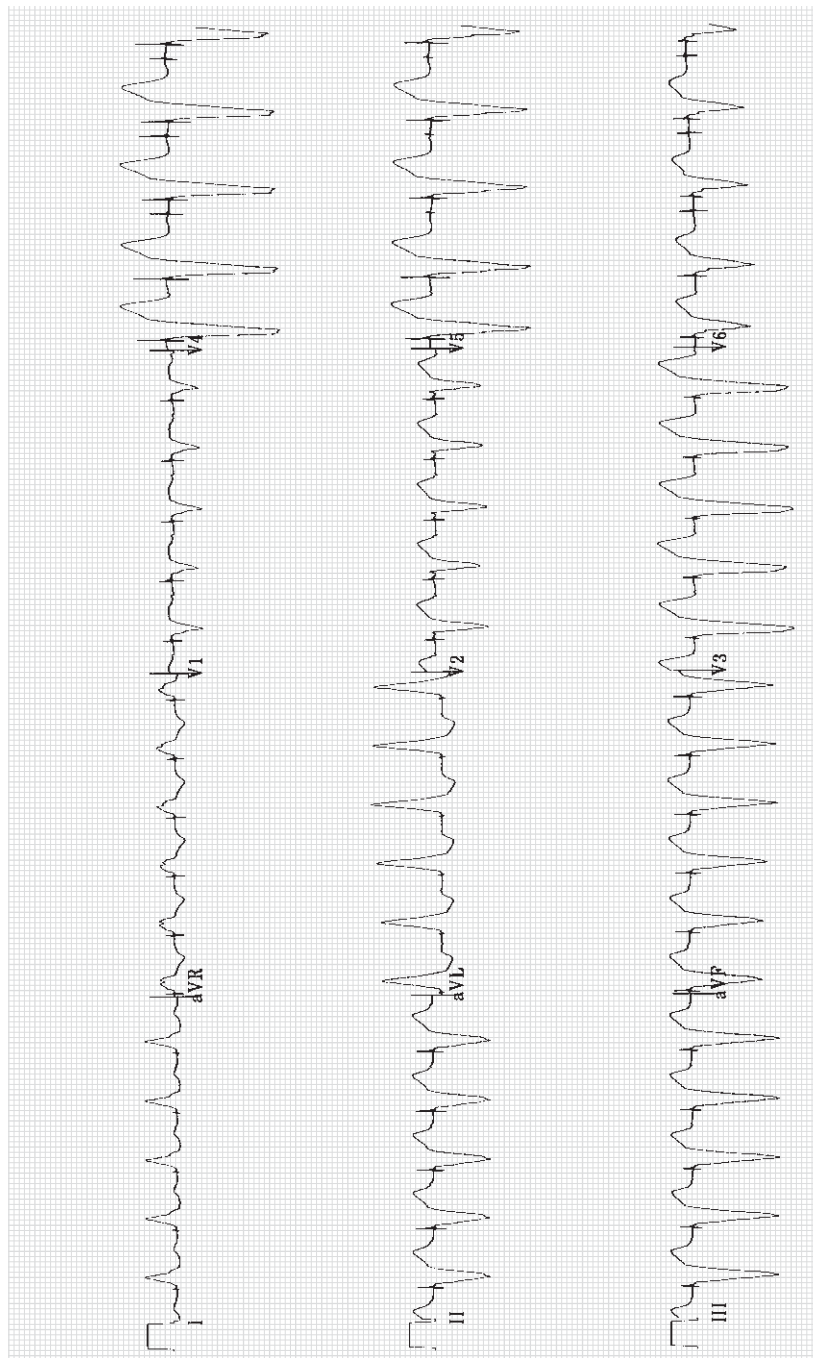


Figure 120 6-year-old girl with dual-chamber pacemaker, programmed DDD, with upper rate limit 150bpm.

Figure 120

- Wide QRS tachycardia at 130 bpm, abruptly drops to 100 bpm
- Ventricular pacing artifacts precede each QRS complex
- During tachycardia, no atrial pacing artifacts are present, suggesting atrial tracking
- During last three beats, AV sequential pacing is seen (pacing both atrium and ventricle)

Pacemaker-mediated tachycardia

- Occurs in setting of DDD pacemaker and intact retrograde atrioventricular node conduction:
 - Ventricular beat conducts retrogradely to atrium via AV node
 - DDD pacemaker senses retrograde atrial activation, and paces the ventricle after programmed AV delay
- Programmed upper pacing rate limit will not be exceeded
- Causes symptoms of tachycardia and leads to premature pacemaker battery depletion

Appendix 1: Age-related normal ECG values in children

Age	Heart Rate bpm	QRS axis Degrees	PR Interval msec	Q III mm	Q V6 mm	R V1 mm	S V1 mm	R/S V1	R V6 mm	S V6 mm	R/S V6	SV1 + RV6 mm	(R + S) V4 mm
<1 day	94–155 (122)	58–168 (+135)	79–160 (107)	5	2	5–27 (14)	0.5–23 (9)	0.2–9.8 (2.3)	0–12 (5)	0.2–10 (4)	0.5–9 (2.5)	2–27 (13)	12–52 (32)
1–3 days	91–158 (124)	65–171 (+134)	81–139 (108)	5	2	5–27 (15)	0.5–21 (10)	0.2–6.0 (2.0)	0.1–12 (5)	0.2–10 (3)	0.5–11 (3)	2–28 (14)	17–53 (33)
3–7 days	90–166 (128)	76–168 (+133)	75–137 (104)	5	3	3–25 (13)	0.5–17 (7)	0.2–9.8 (2.8)	0.5–12 (5)	0.4–10 (4)	0.5–10 (2.5)	2–25 (12)	13–48 (31)
7–30 days	106–182 (148)	65–159 (110)	73–138 (101)	4	3	3–22 (11)	0.5–12 (4)	1.0–7.0 (2.9)	3–17 (8)	0.2–10 (3)	0.5–12 (4)	3–22 (12)	15–48 (31)
1–3 months	120–179 (149)	31–115 (75)	73–130 (98)	5	3	3–19 (10)	0.5–13 (5)	0.3–7.5 (2.3)	5–22 (12)	0.3–7 (3)	0.5–12 (4.5)	6–29 (17)	22–58 (36)
3–6 months	105–185 (142)	7–105 (60)	74–145 (106)	7	3	3–20 (10)	0.5–17 (6)	0.2–6.0 (2.4)	6–23 (14)	0.2–10 (3)	0.5–18 (6.5)	7–35 (19)	21–58 (38)
6–12 months	107–168 (132)	7–98 (54)	73–156 (156)	6	3	2–20 (9)	0.5–18 (7)	0.1–3.9 (1.8)	6–23 (13)	0.2–8 (2)	0.5–22 (8)	7–33 (19)	21–50 (34)
1–3 years	90–151 (119)	8–100 (55)	82–148 (114)	5	3	3–18 (9)	1–21 (9)	0.1–4.2 (1.4)	6–23 (14)	0.1–7 (2)	0.5–28 (9.5)	7–38 (22)	17–48 (33)
3–5 years	73–137 (108)	7–104 (55)	85–161 (118)	4	3	2–18 (8)	2–22 (10)	0–2.8 (0.9)	9–25 (15)	0.1–6 (2)	0.8–30 (11)	13–42 (25)	17–52 (35)
5–8 years	65–133 (100)	10–140 (66)	90–164 (124)	3	5	1–13 (7)	3–24 (12)	0–2.0 (0.8)	9–27 (17)	0.1–4 (1)	1–30 (12)	13–47 (28)	20–53 (36)
8–12 years	63–129 (92)	9–115 (61)	87–171 (128)	3	3	0.5–10 (6)	3–26 (12)	0–1.9 (0.6)	10–26 (17)	0.0–4 (1)	2–33 (14)	15–45 (28)	21–50 (35)
12–16 years	66–120 (86)	11–133 (58)	92–175 (135)	3	3	0.5–10 (5)	3–22 (11)	0–1.8 (0.5)	7–23 (15)	0–4 (1)	2–39 (15)	11–42 (25)	12–49 (29)

ECG normal ranges (2–98%) and mean (in parentheses) from Davignon *et al.* (1979).^a

Appendix 2: Criteria for distinguishing VT from SVT

Wide complex tachycardia: criteria favoring VT

Antunes–Brugada stepwise approach:

Absence of RS complex in precordial leads

R to S interval >100 msec in one precordial lead

More QRS complexes than P waves

Morphology criteria for VT present in V1 and V6

Morphological criteria favoring VT

RBBB morphology:

Triphasic pattern of QRS

Left axis deviation

R:S ratio <1 in V6

LBBB morphology:

qR or QS in V6

R wave in V1 or V2 >30 msec

Any Q wave in V6

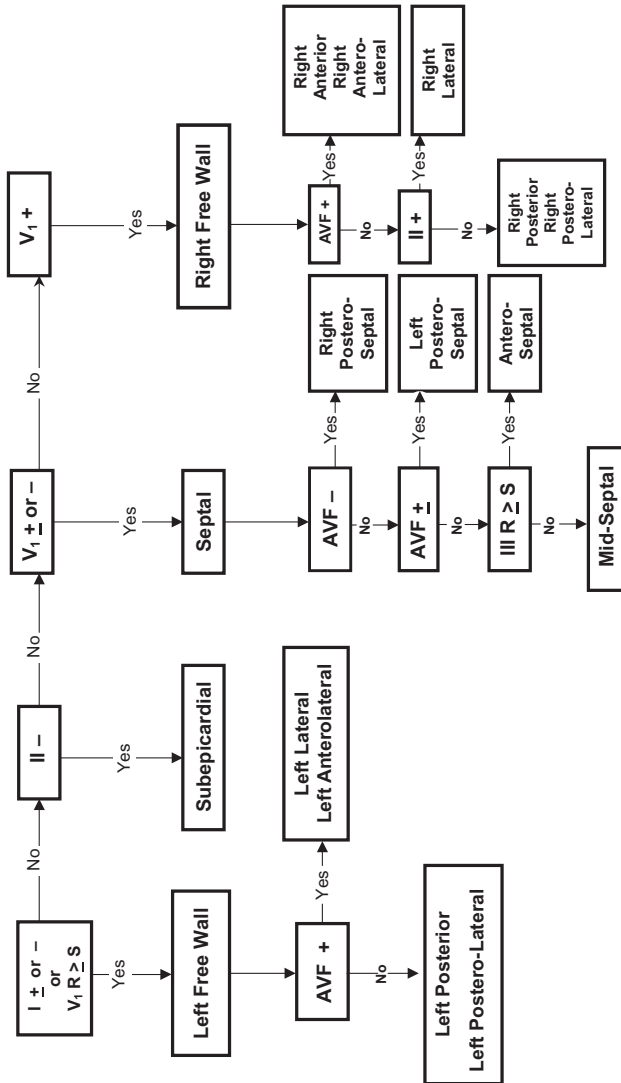
R to nadir S duration >60 msec in V1 or V2

Notching on downstroke of S wave in V1 or V2

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1. Tipler MA. Usefulness of the electrocardiogram in diagnosing mechanisms of tachycardia. *Pediatr Cardiol* 2000;**21**:516–521.
2. Antunes E, Brugada J, Steurer G, Andries E, Brugada P. The differential diagnosis of a regular tachycardia with a wide QRS complex on the 12 lead ECG. *Pacing Clin Electrophysiol* 1994;**17**:1515–1522.
3. Brugada P, Brugada J, Mont L, Smeets J, Andries EW. A new approach to the differential diagnosis of a regular tachycardia with a wide QRS complex. *Circulation* 1991;**83**:1649–1659.

Appendix 3: Location of accessory atrioventricular connection using initial delta wave polarity



Reference

- Arruda MS, McClelland JH, Wang X, Beckman KJ, Widman LE, Gonzalez MD, Nakagawa H, Lazzara R, Jackman WM. Development and validation of an ECG algorithm for identifying accessory pathway ablation site in Wolff-Parkinson-White syndrome. *J Cardiovasc Electrophysiol* 1998;9:2-12.

Appendix 4: Indications for pacing in childhood

Class I

- Sinus bradycardia correlated with symptoms. Note: Numerical definition of bradycardia varies with patient's age and expected heart rate
- Congenital 3° AV block in infant with ventricular rate <55 bpm and otherwise normal heart or a ventricular rate <70 bpm associated with congenital heart disease
- Congenital 3° AV block with a wide QRS escape rhythm or ventricular dysfunction
- Advanced 2° or 3° AV block with bradycardia symptoms, congestive heart failure, or low cardiac output
- Postoperative advanced 2° or 3° AV block that is not expected to resolve or persists at least 7 days after cardiac surgery
- Sustained pause-dependent ventricular tachycardia, with or without prolonged QT on routine ECG, in which efficacy of pacing is thoroughly documented

Class IIa

- Asymptomatic sinus bradycardia in child with complex congenital heart disease and resting heart rate <35 bpm or pauses in ventricular rate >3 sec
- Bradycardia-tachycardia syndrome with need for long-term antiarrhythmic treatment other than digitalis

- Congenital 3° AV block beyond the 1st year of life with an average heart rate <50 bpm or abrupt pauses in ventricular rate that are two or three times the basic cycle length
- Long QT syndrome with 2:1 AV block or 3° AV block

Class IIb

- Asymptomatic sinus bradycardia in adolescent with congenital heart disease with resting heart rate <35 bpm or pauses in ventricular rate >3 sec
- Congenital 3° AV block in asymptomatic neonate, child, or adolescent with an acceptable rate, narrow QRS complex, and normal ventricular function
- Transient postoperative 3° AV block that reverts to sinus rhythm with residual bifascicular block

Class III

- Asymptomatic sinus bradycardia in adolescent with longest RR interval <3 sec and minimum heart rate >40 bpm
- Transient postoperative AV block with return of normal conduction within 7 days
- Asymptomatic postoperative bifascicular block with or without 1° AV block
- Asymptomatic type I 2° AV block

Reference

Gregoratos G, Abrams J, Epstein AE, Freedman RA, Hayes DL, Hlatky MA, *et al.* ACC/AHA/NASPE 2002 guideline update for implantation of cardiac pacemakers and antiarrhythmia devices—summary article: a report of the American College of Cardiology/American Heart Association Task Force on Practice Guidelines (ACC/AHA/NASPE Committee to Update the 1998 Pacemaker Guidelines). *J Am Coll Cardiol* 2002;**40**:1703–1719.

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